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The Manitoba Medical Review

31 MARCH, 1951 No. 3

MEDICINE

he Present Epidemic of Influenza

Adamson, M.D., M.R.C.P. (Ed.), F.R.C.P. (C)

e present epidemic in England is being fol- with intense interest. There can be no that it will spread to this country; no amount arantine could possibly fence it in. Exactly it will come is impossible to estimate. In 918-19 epidemic, the peak in Canada was t a year later than that in Britain. Also, one t predict the extent of the epidemic; that will y depend on imponderable immunological nstances. Also, the severity cannot be pre- l. In spite of the impression one might get our newspapers, authentic medical reports te that the disease carries a low mortality. ver, one can never trust a virus and virulence change for better or for worse during a emic.

is imperative that any cases of influenza that ır should be carefully studied in order to ify the virus. This can be done only if cases eported early and are subjected to the investi- n prescribed by the Health Department. In connection your attention is directed to a ment by Dr. G. B. Leyton, Provincial Bac- logist, appearing in this issue.

Identification of Influenza Virus

G. B. Leyton, M.D.
Provincial Bacteriologist

he Federal Laboratory at Ottawa and the Pro- al Laboratory are interested in isolating the from the cases of epidemic influenza. The incial Laboratory has a few special containers he collection of throat washings, and is also ested in receiving paired specimens of blood, first obtained early in the disease and the d during convalescence. When small epi- cs break out, it would be much appreciated if bers of the medical profession would ring up Provincial Laboratory who will then, if the sounds suitable, provide the necessary appa- for taking the specimen. Only a few mens are required from each epidemic area. he disease in Britain is caused by "Virus 'A' e." This is said to be a strain of virus not previously in this country. The virus vac- available give partial immunity to the strains which they are made, but there is no inty that they would be of any value if they not contain the strains infecting the patient.

Influenza*

J. D. Adamson, M.D., M.R.C.P. (Ed.), F.R.C.P. (C)

Introduction

During the past fifty years in civilized countries, perennial devastating epidemics have almost disappeared. The exanthemata have become rare and attenuated; widespread outbreaks due to pyogenic organisms are infrequent, readily recognized and effectively treated. Unfortunately we cannot be so complacent about virus infections; indeed our position appears to be becoming worse†.

†See Chart 2, page 140.

New epidemics are appearing the world over. In Manitoba the situation would appear to be more virulent than in most parts of the world. In the period from 1919 to 1923 there was a very high incidence of encephalitis lethargica (von Economo encephalitis) which carried a high mortality and left an appalling legacy of Parkinsonism. In 1918-19 the community suffered its share of the most malignant influenza pandemic of modern times. In 1941 we had the distinction of being the centre of an epidemic of Western Equine encephalitis coincident with a large poliomyelitis attack. Besides these clear-cut outbreaks there have been regular epidemics and sporadic cases of influenza, colds, poliomyelitis and other unclassified virus infections.

The memory of these epidemics provides food for serious thought and sombre speculation. Evidently new viruses, or old viruses with enhanced virulence, can run riot at any time, and no one can predict when and how they will strike. We are almost completely without facilities for their precise diagnosis and no specific treatment has been discovered for any one of them. Each one takes us by surprise and finds us completely helpless. Fortunately in the past the morbidity has not been overwhelming and the total mortality has not been great. This is entirely a matter of chance and certainly not related to any effort on our part. Picture what could happen if the next virus that goes berserk is slightly more virulent than any of these that have so far surprised us. It requires very little imagination to create a state of apprehension not less than that associated with the future of atomic energy. In view of these facts and these possibilities, it is not an exaggeration to say that the clinical laboratory study of virus is the most important epidemiological problem of our day.

*From the Department of Medicine, University of Manitoba, Winnipeg, Man.

Influenza is the most important of all the virus infections because it is so common and because from time to time it becomes pandemic and produces an enormous total mortality. Its study is particularly indicated at this time because definite serological assistance in diagnosis is becoming available, prophylactic measures are promising and treatment with new antibiotics may be effective. Primarily it is a generalized infection and not a "respiratory disease"; but since the only real danger lies in respiratory complications and it is these that demand treatment, it is appropriate to consider it here.

History

The term "influenza" has been in use since the fourteenth century and originated in the belief that epidemics were due to malign astrological influences ("ab oculta coeli influenza"[1]). Since then the word has been applied to a great variety of epidemics and pandemics in various parts of the world. In fact, any febrile epidemic which did not conform to the pattern of a recognized infection of the period was likely to be called "influenza." Many of the "plagues" and "pestilences" of ancient history are sometimes attributed to influenza but often on very meagre evidence. The term has been particularly applied to conditions in which respiratory, cerebral or gastro-intestinal complications were common. The clinical syndrome that is now called by this name has been clearly described as far back as authentic medical history can be followed and it has unquestionably been the cause of innumerable epidemics and pandemics. The scope of the term has gradually been reduced as other specific infections have been recognized. As commonly used, it still includes a great variety of infections and even in the strictest sense is not truly specific but is due to an indefinite number of viruses.

Some extracts from classical descriptions are of interest, and will give a picture of the gradual development of the present belief. In "The Modern Practice of Physic, exhibiting the Characters, Causes, Symptoms, Prognostic, Morbid Appearances, and Improved Method of Treating, the Diseases of All Climates," third edition, London, 1811, Dr. Robert Thomas of Salisbury, writes:

"The catarrhal fever known by the name of Infuenza, which prevailed so universally in most parts of this kingdom in 1803, as well as in France, where it was called La-gripe, first shewed itself in London towards the latter end of the month of February, when a damp and mild state of the atmosphere had succeeded to severe cold, and when this again had been followed towards the beginning of March by frost and keen easterly winds.

"Like preceding epidemics of the same this disease exhibited various degrees of affection, having been in some instances so as not to incapacitate persons from following ordinary occupations and pursuits, and scar require the aid of medicine; while in othe attack was of so severe a nature as to en life, and even to destroy it."

. . . . . .

"It was generally preceded by chilline shiverings, which were succeeded by some of heat, pains in the head, a discharge from th and nostrils, severe sneezing, hoarseness cough. . . . Some patients complained of pa the shoulders and limbs, very much rese chronic rheumatism, and there were instan which the difficulty of breathing might be, attributed to a similar affection of the inte muscles."

. . . . . .

"By some physicans the disease was su to be contagious; by others not so; indeed i and rapid spread made many suspect som generally prevailing cause in the atmosph alone capable of accounting for its extensi speedy diffusion. It arose, probably, at firs a peculiar state of the atmosphere, like oth demics, and was afterwards kept up and gated by contagion."

The following extracts are taken from "Lectures on the Practice of Medicine," by J. Graves, M.D., F.R.S., 2nd edition, Londo

". . . when an epidemic like influenza its appearance, everything comes under fluence almost simultaneously, and, like it overshadows the whole country in the s a few weeks. Such was the course of t demics of 1837 and 1847, and so it was w influenza of 1782, which travelled from tre and left traces of its ravages in almost quarter of the globe."

. . . . . .

"Several epidemics of this descriptio been distinctly recorded in the eighteenth c viz., in 1708, 1712, 1728, 1733, 1743, 1758, 17 1775, 1782, 1788, 1789; while in the portion nineteenth century already elapsed five in have occurred, viz., in 1803, 1831, 1833, 1 1847."

. . . . . .

". . . we may conclude that in Dubli more than four thousand people died of fluenza of 1837, not taking into account the number who, although they got over the diate attack of the epidemic, sank aft under various diseases, of which influenza the foundation."

. . . . . .

"It would conduce greatly to the adva medical science, if a brief and accurate his

[1] Hamer, W. H.: Influenza, edited by F. G. Crookshank, Heinemann, London, 1922.

t to posterity, of the character, symptoms, thological phenomena, and treatment of every idemic. Such a record would prove a guide and con to the practitioners of future ages—would able them to draw important comparisons be- een the existing and the past—and thus arrive a more fixed and available knowledge of the ure and habits of epidemic complaints."

. . . . . .

". . . in influenza or bronchitis you seldom e true pneumonic inflammation."

In Osler's first edition (1892), from which the owing extracts are taken, the term still in- ded a wide variety of infections. Bacteriology then in its vigorous infancy and specific anisms were sought in every fever. The word us" at that time included all pathogenic anisms. Osler's attitude to influenza was evi- tly largely formed by his experience in the t pandemic of 1889-90.

"**Definition.** An infectious disease character- by great prostration and often catarrh of the ous membranes, particularly the respiratory gastro-intestinal. There is a marked liability erious complications, particularly pneumonia."

. . . . . .

"**Etiology.** . . . While some authorities hold the affection is due to a miasmatic material ne atmosphere, others probably more correctly that it is due to a specific virus of the most nse infectiveness. . . . The pus organisms and **diplococcus pneumoniae** have been found nest, but these are widespread organisms and probably not associated in a causative manner the disease."

. . . . . .

"**Symptoms.** . . . A striking feature is the re nervous manifestation at the outset, the ache, pain in the back and legs, and a general ess as if bruised or beaten. With the excep- of dengue and smallpox there is no affection hich these symptoms are more pronounced. Sometimes the symptoms may at first be re and the pneumonia atypical. . . .

The diagnosis of the disease offers no diffi- es when it occurs in epidemic form. Coryza t always present, and the symptoms may be of general fever with great prostration."

great variety of complications were at- ted by Osler to influenza, e.g., nephritis, ngitis, brain abscess, peripheral neuritis, lung ss, pleurisy and empyema; it is evident from that primary and secondary pyogenic infec- were still causing confusion.

1918 when Osler's last edition was published, nfluenza bacillus (Pfeiffer) was generally ac- d as the cause of influenza as the following cts show:

"**Definition.** A pandemic disease, appearing at irregular intervals, characterized by extraordinary rapidity of extension and the large number of people attacked. Following the pandemic there are, as a rule, for several years endemic, epidemic, or sporadic outbreaks in different regions. Clinically, the disease has protean aspects, but a special tendency to attack the respiratory mucous membranes. A special organism, **Bacillus influenzae,** is found."

. . . . . .

"**Bacteriology.** In 1892 Pfeiffer isolated a bacillus from the nasal and bronchial secretions, which is recognized as the cause of the disease."

. . . . . .

"**Diagnosis.** During a pandemic the cases offer but slight difficulty. The profoundness of the prostration, out of all proportion to the intensity of the disease, is one of the most characteristic features. In the respiratory form the diagnosis may be made by the bacteriological examination of the sputum, a procedure which should be resorted to early in a suspected epidemic. The differentiation of the various forms has been already sufficiently considered."

The premature acceptance of Pfeiffer's bacillus as the specific cause of influenza has created great confusion which still persists. For example, the specific meningitis caused by Pfeiffer's organism is still called "influenzal meningitis," though it has no relationship to the disease influenza as we now know it.

In 1933 Smith, Andrewes and Laidlaw[2] working in the National Institute for Research at Mill Hill, England, obtained throat washings from influenza patients which, after filtration through a membrane impermeable to bacteria, produced a febrile illness in ferrets. No other susceptible animals were found but the infection could be serially transmitted in ferrets by nasal instillation of nasal secretions. Serum from human influenza convalescents was found to contain antibodies capable of neutralizing the virus of the ferret disease.

This identification of a virus of influenza is the most important event in the history of the disease. It has stimulated a vast amount of research. Two separate viruses, "A" and "B," have been identified by their antibody production. Epidemics due to each of these have been identified and vaccines have been prepared which enhance resistance to infection but do not confer complete immunities. Unfortunately these two viruses do not account for all outbreaks; other related but still unidentified viruses can produce fevers that are clinically indistinguishable from that shown to be due to virus "A" or "B."

[2]Lancet, 2:66, July 8, 1933.

Annual Death Rate in London Per Thousand Living Over a Period of 85 Years

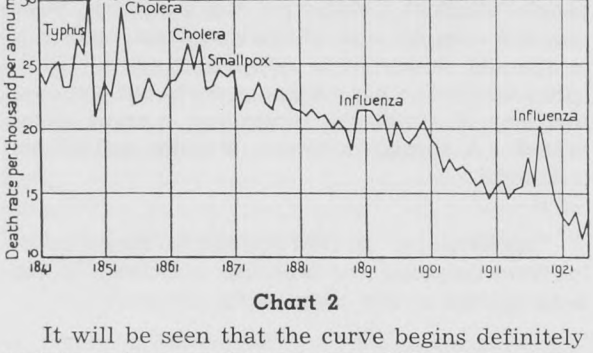

Chart 2

It will be seen that the curve begins definitely to take a downward trend about 1870. It has been falling ever since. It is now less than half of what it was sixty years ago. This fall is largely, though not entirely, due to decrease in infant mortality. Some of the more important epidemics are indicated. Typhus disappears as an important cause of death in the forties and Cholera and Smallpox in the sixties. Since then the deathrate has been considerably modified by Influenza outbreaks[5].

Among other things this chart demonstrates that influenza is the only remaining devastating, pandemic disease of menacing proportions.

Annual Death Rates From Pneumonia and Influenza, and the Range of Monthly Variation*, 1925 to 1949

Weekly Premium-Paying Industrial Business, Metropolitan Life Insurance Company

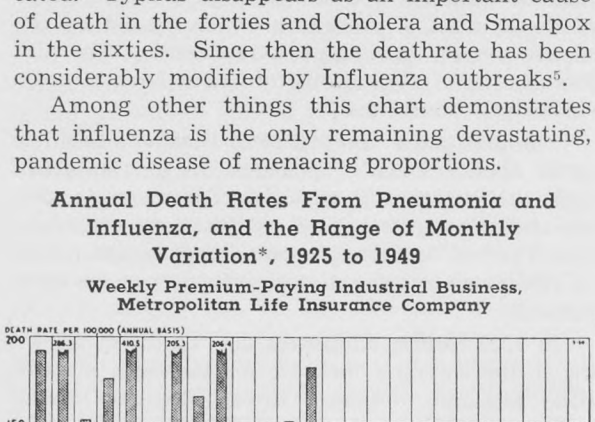

Chart 3

*The annual death rates are shown by the continuous black line. The range from the lowest to the highest monthly death rate within the year is indicated by the height of the vertical shaded bar.

Diagnosis

The discovery of the cause of influenza makes its clinical study and identification more important than formerly. Meticulous clinical investigation has not kept up with laboratory investigat This is regrettable since it is only by bed study that symptoms and signs that may peculiar to each virus will be recognized. Tho epidemics due to both "A" and "B" have proved serologically and others have been pr to be due to neither of these viruses, their cli differentiation is still vague. One gets the pression from identified cases that the disease to "A" is more abrupt in onset, of shorter dura and is followed by fewer complications.

It is often said that a bedside diagnosis influenza cannot be made. This is true, if o speaking of an absolutely infallible diagnosis in Medicine such security in diagnosis is possible. In general, virus influenza can be re nized if sufficient care is taken. It is chiefly careful history of onset that a diagnosis ca made.

The criteria upon which influenza mus suspected are enumerated and discussed.

1. Onset:

This is very abrupt; within a few hours onset the victim is reduced from a normal of health to one of partial or complete prostra

2. Initial Symptoms:

During the first few hours these are a predominantly of a constitutional character. patient is ill all over and cannot indicate any origin of his trouble; that is, he has no symp referable to his nose as in a common cold, n throat as in streptococcal pharyngitis a localization of pain over any particular reg organ.

3. Symptoms:

There are no characteristic symptoms and that cannot occur with the sudden onset of fevers. Diagnosis depends on the groupi symptoms and their sequence. The most s complaints at onset usually are: chilliness rigor), prostration, headache, backache, so "tired" muscles, pain on movement of th and absolute anorexia. In most cases a dry ness of the throat and trachea associated w unproductive cough makes its appearance twenty-four hours. Table I shows the perc incidence of various symptoms during th two days in 68 cases who were carefully foll

Table I

	First day	Secon
Malaise	81%	1
Headache	71%	1
Cough	60%	2
Chills	60%	1
General pains	23%	
Sore throat	24%	2
Nasal discharge	9%	

[5] Singer, Charles: A Short History of Medicine, Oxford at the Clarendon Press, 1928.

4. Physical Findings:

In uncomplicated cases, except for the fever there is nothing to find. Though there may be some complaint of sore throat, examination of the throat is usually negative. Various signs frequently appear in the lungs but these are regarded as complications.

5. Temperature and Pulse:

The temperature rises suddenly to a level that varies widely from case to case but is usually about 102° or 103°. The pulse is relatively slow, often remaining below 100 in spite of high fever. After the first day all symptoms gradually improve and, with the exception of general physical and mental depression, should have passed off within a week.

6. Complications:

Nearly all cases will have some respiratory complications. These will be referred to later.

7. Failure to Respond to Antibiotics:

There is complete lack of response to sulpha drugs and to penicillin. At the moment the value of aureomycin, terramycin, chloromycetin and some other antibiotics is not fully assessed.

8. Leukocyte Count:

There is no consistent or persistent granulocytosis. Frequently there is a leukopoenia but in the first 24 hours the total count may go to 14,000 and there may be a slight shift to the left; this is rare and does not persist.

Differential Diagnosis

Influenza may usually be recognized on the above criteria, during the first or second day. But since many other diseases at their onset may cause almost identical symptoms, every effort should be made to exclude them. The chief confusion arises with the common cold. This is largely a matter of nomenclature; many people still do not attempt to make the differentiation and appear satisfied to group them together under such terms as "la grippe." This is unfortunate, especially since there is no good reason for confusion. They are definite clinical entities and are strikingly contrasted in onset and symptoms. Table II compares some of the features of these two conditions.

Table II

	Influenza	Common Colds
Onset	Sudden	Insidious
Constitutional Symptoms.	Marked— Headache. Fever and chills. Malaise. Prostration. Muscle pains. Anorexia. Vomiting.	Mild— Slight chilliness. Slight malaise. Slight Muscular pains. Very little fever.
Local Symptoms.	Mild or absent— Cough. Sore throat. Retrosternal discomfort.	Marked— Nasal discharge Sore throat Laryngitis. Tracheitis, etc.

The onset of pyogenic infections may sometimes be abrupt and with constitutional effects. In such cases there will usually be symptoms or signs pointing to the site of the infection, e.g., pyelitis, perinephritic abscess and osteomyelitis. In any of these there will always be a leukocytosis.

Occasionally acute pneumonia may be "silent" or "occult"; by which is meant that no localizing signs or symptoms are apparent. Pulmonary tuberculosis or pleurisy may also come on acutely without any convincing localizing indications. In all of these cases the diagnosis may be in doubt for a day or so. Many other febrile illnesses, e.g., typhoid fever, miliary tuberculosis, glandular fever, sub-acute bacterial endocarditis and acute leukaemia, are occasionally at first called influenza. Such errors are nearly always due to an inadequate or inaccurate history of onset.

During non-epidemic periods one is well advised never to dismiss febrile illness with a diagnosis of influenza until it has been under observation for a day or two and other possibilities have been considered. During epidemics there is an added danger of over-diagnosis; during such periods many acute exacerbations of tuberculosis and pneumonia are quite frequently improperly judged to be part of the epidemic.

An absolute diagnosis of influenza may be made only by serological tests. These methods are of no value in the immediate diagnosis of individual cases since they depend on a significant rise in the specific antibody during the illness. It is necessary, therefore to estimate the antibody titre soon after the onset and again soon after the symptoms have disappeared. The method is, however, of great importance in the recognition of epidemics. If the earlier cases can be identified as "A" or "B," vaccination throughout an institution or community may be of definite value.

Complications

A multitude of complications have been ascribed to influenza in the past. Most of these appear to have been due to pyogenic infections and not to virus influenza as we know it. The only common complications are infection of the respiratory tract and of these there is a great variety.

A large proportion of cases in any epidemic have subjective evidence of pharyngitis and tracheitis. These have surprisingly few signs of inflammation on inspection of the throat. Many cases develop all the signs of acute diffuse bronchitis. The incidence of complicating virus pneumonitis varies greatly with different epidemics and possibly with the type of virus.

The above respiratory complications are presumably due to the virus itself and none of them is serious; certainly post influenzal virus pneumonitis is comparatively innocuous and does not account for the enormous mortality that accom-

[2] Adamson, J. D. and R. O. Flett: The Inefficacy of Sulfadidine in Influenza, The Canadian Medical Association Journal, 46: 121-123, 1942.

panies some epidemics. The serious pulmonary complications have usually been attributed to secondary bacterial invaders. Influenza has always had the reputation for "preparing the soil" for pyogenic organisms. In the 1918-19 epidemic there was a high mortality from "secondary" acute fulminating broncho-pneumonia. In the light of present knowledge there must be some doubt that such a symbiosis or sequence actually did occur. At the time no virus had ever been identified and even the bacteria involved was not agreed upon. It is possible that no virus was implicated or that the virus was quite different from those that are now considered to cause influenza. Also, since 1919 there has been no marked tendency for influenza to pursue the course that was so common then. In spite of this the fear of secondary invaders still persists and cannot be dismissed completely. It may be that such explosive and devastating epidemics depend on the chance coincidence of appropriate strains of virus and streptococci.

Gastrointestinal complications (vomiting, abdominal cramps, looseness of bowels) are said to be common in some epidemics and the term "stomach flu" is popular. There seems no doubt that there is a gastroenteritis due to a virus but it has not been identified nor has any epidemic of gastroenteritis been shown to be caused by "A" or "B." It seems likely that these symptoms are no more common in proven influenza than in any acute fever and that they depend on individual susceptibility.

Treatment

Since there is no specific treatment, the general measures appropriate to any acute fever are prescribed. In view of the possibility of secondary pyogenic infection penicillin may be given. It appears probable that at least some influenza-like viruses are inhibited by aureomycin, chloromycetin and terramycin but no convincing evidence of this has been produced.

Prophylaxis

It has been well established that a degree of protection may be established against virus "A" or "B" by vaccination. The American army has established a commission to investigate influenza; a summary of the preliminary report follows:

"The influence of subcutaneous inoculation of a concentrated inactivated vaccine on the incidence of clinical influenza in a series of Army Specialized Training Program units comprising approximately 12,500 men was studied during the recent epidemic of influenza A. Vaccination done shortly before or even after the onset of the epidemic was found to exert a protective effect with a total attack rate of 2.22 per cent among the 6,263 vaccinated and 7.11 per cent among the 6,211 controls, a ratio of 1 to 3.2. The influence of vaccine was most clea evident at the height of the epidemic prevalen The duration of the effect has not been de mined[4]."

Since the above report, other vaccines wit wider range have been elaborated. Since ther a variety of strains of both "A" and "B" and s other viruses, still unidentified, can produce Influenzal Syndrome, it is always difficult to dict the efficacy of vaccination.

[4]Report of Influenza Commission, School of P Health, University of Michigan, Ann Arbor, Mich. J.A 124:14, 985, April 1, 1944.

Addendum

After this article was written, one case of nite clinical influenza has been admitted to Lodge Hospital. This youth was stationed Gimli where there was a large recent influ recruits from England. The identification of virus has not yet been completed but there se little doubt that it is in fact the continental demic.

Influenza

Letter to All Physicians and Hospital Staffs:

You have no doubt been reading in the ne papers and hearing over the radio about influenza epidemics in Europe. There has been an outbreak in Newfoundland.

Information received from the Departmen National Health and Welfare at Ottawa, quo reports from the World Health Organization, the effect that complications are rare and mortality low, and in neither spread nor sev can this outbreak be compared with those o ring in the first part of this century. Almos deaths have been of persons in the older groups. Antibiotics have proved successful ag secondary invaders. The causative virus "A" prime—of a type not isolated heretofo Canada. Any vaccines available here therefore be of no use as a preventive agains type of influenza.

This epidemic may spread to Canada be of the large number of persons travelling Europe.

Influenza is a notifiable disease under the lic Health Act of Manitoba but because extreme variability is poorly notified. We ask you to report all Typical Cases of infl to the Department on our usual notification If you are too busy to fill out cards kindly me a call by telephone or drop me a line me know that you are seeing cases of infl Our Department and the National Departme anxious to be informed so that any nec action may be taken.

Through the Dominion Laboratory of Hygiene at Ottawa we hope to recover the virus as a matter of identification of type and for further study.

If you have a typical case of influenza in the early stages get in touch with Dr. G. B. Leyton, Director of Provincial Laboratory, at the Medical College, phone 27 413. It must be remembered that these specimens entail a great deal of special laboratory work so only a few will be taken in each province and those only from typical cases. This procedure is of no value in diagnosis, except in retrospect, and is not intended as a diagnostic procedure.

Please keep these facts in mind and if you have anything to report, write to me at 320 Sherbrook Street, Winnipeg (or phone 37 131).

Maxwell Bowman, M.D., D.P.H.,
Director.

The Middle Lobe Syndrome*

W. Carleton Whiteside, M.D.

In recent years greater knowledge of the anatomy and pathology of the bronchial tree has been established. A number of lesions now recognized as clinical entities were once grouped under the general terms of, "bronchitis" or "congestion of the lung."

The middle lobe syndrome, or chronic atelectasis and pneumonitis of the right middle lobe is one of the new recognized clinical entities deftly plucked from the "bronchetic heap" of pulmonary diagnoses. Much credit is due to Mr. R. C. Brock of London, for placing his research findings in the literature and in his teaching. Many cases of middle lobe syndrome have since been reported. The author's first case was treated twelve years ago, although it was not recognized under this diagnosis at that time.

The essential pathological features of this condition consists of a bronchial obstruction of the middle lobe by the peribronchial lymph nodes following an infective process. This is followed by atelectasis, pneumonitis and later by fibrosis of the lobe. The peculiar arrangement of the nodes and the position of this bronchus appears to be an added factor causing such a lesion. Mr. Brock considers that a number of the lymph nodes are tuberculosis in origin which become calcified and erode the bronchus causing ulceration, haemorrhages and broncioliths and further obstruction. Cultures from these nodes would appear to substantiate Mr. Brock's theory.

The cardinal symptoms of the middle lobe syndrome consists of a chronic cough, at times nonproductive, a pleuritic type of pain in the right chest, haemoptosis is common, dyspnoea due to atelectasis and a wheeze is noticed, this being due to a partial blockage of the bronchus to the middle lobe.

The cough is generally worse in the dorsal recumbent position. This could be due to "an emptying" of the bronchus into the dorsal lobe bronchus while in this position as the middle lobe bronchus lies in a dorso-ventral position and the dorsal lobe bronchus being a "trigger cough" bronchus is readily stimulated.

In the middle lobe syndrome a history of previous "pneumonias" is common. A low grade fever persists after the clinical features have subsided. A persistence of symptoms from five months to twenty years have been recorded.

In order that the diagnosis be made the lesion must be thought of in patients who have the above symptoms. A right lateral film is essential together with the usual posterior-anterior exposure, in order to visualize the right middle lobe properly.

By means of the bronchoscope and bronchograms the bronchus will be found to be blocked or partially closed. The bronchioles are closer together due to the fibrosis, the interlobar fissures are likewise close together, the bronchi of the upper lobe may be distorted due to the contracted state of the middle lobe. The triangular—atelectatic—lobe will be seen by the above procedures as well as the narrowing of the intercostal spaces, the raised diaphragm and the cardiac shift.

The most important lesion from which the middle lobe syndromes should be diagnosed is primary carcinoma of this bronchus. This can be ruled out by a bronchoscopic biopsy.

The lesion is surprisingly common when looked for and relief from symptoms by removal of the involved lobe gives excellent results. If the atelectasis of the middle lobe has been for a long period then bronchiectasis becomes an added complication and cough is more productive. Treatment by removal is most gratifying.

The middle lobe syndrome is a common clinical entity responsible for many of the illnesses now masquerading under the term of "chronic bronchitis" or "recurrent flu." Its diagnosis should be made as a clinical entity.

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Assessment of Pulmonary Function by Clinical and Laboratory Methods*†

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Respiration should not be regarded as an isolated function of the lungs, but rather as the combination of different processes interacting with one another to serve the metabolism of the tissue cells in the body. These processes comprise: (1) The ventilation or actual movement of air in and out of the lungs; this is purely mechanical performed by the chest cage, tracheo-bronchial tree and the diaphragm; (2) The respiratory aspect, which consists of the gaseous exchange of oxygen and carbon dioxide into the blood through the alveolar walls (largely a physio-chemical process); and (3) The gaseous exchange that goes on between the blood and the tissue cells during metabolism.

Respiration is, therefore, a respiratory-circulatory mechanism, in which a balance exists between the lungs and the circulation, so finely adjusted as to produce an immediate response to any change in metabolic levels. The hormones of respiration are carried by the circulation to the respiratory centre in the medulla, which when stimulated results in increased pulmonary ventilation. The increased negative intrapleural pressure which is produced causes a greater flow of blood into the right side of the heart from the great veins, as well as aiding the coronary circulation. The increase in metabolic requirements that takes place with exercise, after taking food and in any febrile state, produces an increase in the circulatory blood-flow by a rise in the heart-rate and cardiac output, an increase in the blood pressure and an alteration in the calibre of the blood vessels throughout the body. The higher rate of circulation of blood through the pulmonary vessels results in less blood being in actual contact with the alveoli during a given time. In order that this more rapidly flowing blood may still become saturated with oxygen and its carbon dioxide content adequately dealt with, the ventilation of the lungs is altered by increasing the rate and depth of respirations. This reserve power of the respiratory processes is comparatively great, and the metabolism can be increased many times in normal individuals without an inadequate supply of oxygen or an accumulation of carbon dioxide developing in the tissues.

Clinical Assessment

From the clinical point of view, pulmonary function can be assessed by the presence of dyspnea and cyanosis. Dyspnea is the symptom produced by impairment of the ventilatory function, whereas cyanosis occurs if there is reduction in the degree of gaseous interchange through t alveolar walls. The ventilatory and respirat mechanisms are closely inter-related. Clinica however, it is not unusual to find cases of alm pure ventilatory insufficiency, whereas cases pure respiratory insufficiency are very rare. Usu ly ventilatory insufficiency occurs in any disea process which affects the function of the p monary parenchyma, bronchial tree, pleura, d phragm, or ribs, and this will eventually lead a reduction in the efficiency of the respirat factor. Conversely, impairment of the respirat function usually causes stimulation of the resp tory centre, resulting in increased pulmon ventilation.

The degree of dyspnea is an important facto the clinical assessment of decrease in ventila function. If the dyspnea only occurs with exer slight ventilatory insufficiency is present. dyspnea is present at rest, serious impairmen ventilation is present. It is only when dysp at rest has been present for a considerable pe of time that cyanosis may develop. For pract purposes, the degree of pulmonary insuffici can be assessed by studying the ventilatory f tion alone. Inadequate aeration of the blood the lungs is ordinarily tested by measuring oxygen saturation of the arterial blood, a me which is still too difficult for routine use.

Besides the subjective sensation of dyspnea the presence of cyanosis, certain other cli methods are used to assess the degree of pulmo insufficiency. These consist of: the position o patient in bed as well as the number of pil used; the use of the accessory muscles of res tion and alae nasi; the extent of the disease cess in the lungs as shown by the physical fin and roentgenological examination; and final the fluoroscopic demonstration of the degr movement of the diaphragm and ribs.

Fluoroscopy of the chest is of great val estimating the pulmonary function of each The ribs and diaphragm are watched during mum inspiration and expiration, the degr movement being compared on the two side the angle through which the ribs move i than nine degrees, then restriction of chest e sion on that side is present. By recording th centage of rib motion and diaphragmatic exc for each individual lung and adding the perce figures for each side, it is possible to estima ratio of ventilation carried out by each lung an example, if on the right side it is esti that the ribs have 100% movement and th phragm has 100% excursion, making a to 200%, whereas on the left side the ribs only 25% and the diaphragm 25%, making a to 50%, then the right lung is performing 80% total ventilatory functions.

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Laboratory Assessment

The degree of pulmonary insufficiency as determined by the clinical and radiological methods is at best only a very gross estimation. More accurate and scientific methods of assessment must be found, and studies in this field have been in progress for many years. Accurate assessment of pulmonary function has become more important in the past decade due to the rapid development of thoracic surgery, and the fact that more cases of pulmonary fibrosis due to industrial disease are being granted compensation.

From the time of Hutchinson, who first introduced spirometry and the study of vital capacity in 1846, these investigations have been carried on to the present day. It was largely through the work of Hutchinson and Panum in 1868 that the various subdivisions of lung volume that are in use today were devised. It was Bohr in 1907 who was first able to estimate the residual air by the use of the principle of the mixture of gases. This discovery made it possible for the first time to estimate all the components of the lung volume. These components, however, only give the anatomical or static measurements of the lung and are not very helpful in elucidating the degree of pulmonary insufficiency. It is only the study of the kinetic or functional activity of the lungs which has been carried out in recent years that has enabled methods to be devised which allow the clinician to assess with a fair degree of accuracy the amount of pulmonary insufficiency in a given patient. The use of tracings produced by means of the spirometer is of especial value. It provides objective records which can be used for comparison as the pulmonary disease progresses as well as indicating the results of any therapeutic procedure.

The use of the spirographic method fulfills certain principles that are necessary if a test is to be of practical value. The apparatus itself must be light and portable, so that the apparatus can be brought to the patient's bedside if necessary and be used by the patient in the recumbent position. The discomfort produced on the patient must be no greater than that caused by the use of the nose-clip and the mouth-piece. The procedures used must be simple and easily understood by the patient. The gas inhaled must consist only of atmospheric air, as the introduction of a foreign gas such as carbon dioxide or hydrogen may influence the respiration.

The apparatus that is used is a recording spirometer which is an adaptation of the Benedict-Roth apparatus used for the estimation of the basal metabolic rate. This is a closed circuit, consisting of a nine-litre capacity gasometer, a glass flask containing a solution of caustic potash, a small pump driven by an electric motor, and a three-way tap connected to the patient by means of a rubber mouth-piece. The movement of the gasometer as the patient inspires and expires is recorded in ink by means of a ventilometer which reduces the respiratory excursions by twenty-five times. The record is made on a kymograph which has two speeds, one which is slow, similar to the one used in basal metabolic readings, and a speed which is five times as rapid. As the patient breathes in and out of the closed-circuit, the carbon dioxide in the air is absorbed by the caustic potash. By the constant addition of oxygen to the spirometer, the recording line on the kymograph can be kept level. Free circulation of the air in the closed-circuit is assured by the electric pump, thus eliminating any dead space in the apparatus.

The patient is seated comfortably in a chair, or if he is confined to bed, the most comfortable position is found, and he rests quietly for at least fifteen minutes. The nose-clip is attached, the mouth-piece is placed in position, and the patient begins to breathe through the closed circuit of the apparatus. In order to accustom the patient to the apparatus and to avoid uneven breathing, quiet inspiration and expiration is carried on for five minutes. This is the "tidal" breathing, and the volume of air taken in with each breath is called the "tidal volume."

The equilibrium level that is used for estimating the beginning of the various components of the lung volume is the point at the end of a quiet expiration. Starting from this equilibrium level, the "complementary volume" is estimated by asking the patient to take in as deep an inspiration as possible. After a short period of quiet breathing, the "vital capacity" is determined by measuring the greatest expiration that can take place after inspiring the "complementary volume" of air. "Vital capacity" consists of the sum of the reserve air, tidal air and complementary air. The air that still remains in the lungs after a maximum expiratory effort is called the "residual air," and this cannot be measured by spirographic methods. At the end of an ordinary expiration, the volume of air that remains in the lungs is known as the "functional residual air," consisting of the sum of the "reserve air" and the "residual air."

These measurements do not take into account the "dead space," which is the inspired air that does not take part in the respiratory exchange of gases. The "dead space" is a physiological, not an anatomical, conception, and the methods that are available for the estimation of the size of this space are too complicated and difficult for routine use. The results, besides, are too unreliable. The usual figure given for this is 120 to 140 cc., but it is increased a further 100 cc. if the respirations are deep. As it is only the amount of air that actually reaches the alveoli, or "alveolar volume" that is of importance (being the only air which

takes part in the respiratory exchange of gases), this could be estimated if the "dead space" could be accurately determined. As an arbitrary figure has to be used for the "dead space," the figures obtained are not reliable.

By the use of a time-marker on the kymograph, the rate of tidal respiration is accurately measured. By noting the volume of air expired over a given period of time, usually one minute, together with the respiratory rate, the "minute-volume" of ventilation, as well as the volume of air expired in a single respiration, can be measured. The normal figures for "minute-volume" vary considerably with the size of the individual. Even in the same subject this will vary, depending on whether his respirations are slow and deep or shallow and rapid. The average figure given for "minute volume" in an individual at rest is between 4 and 6 litres. The tracing will also show the steepness as well as the amplitude of both inspiration and expiration. This, too, will vary in different individuals, depending on the rate and volume of air inspired over a period of one minute. No matter what the volume is, the time ratio between inspiration and expiration remains approximately constant at 1:1.1. The tracing will also show any variations, such as Cheyne-Stokes respirations, the sighing respirations of the neurotic and the shallow respirations of the malingerer.

The measurement of "vital capacity" indicates the limit to which respiration can be increased in that individual. During ordinary breathing, while the patient is at rest, use is made only of a small portion of the vital capacity, usually about 10%. With hyperpnea, such as occurs with muscular exertion, up to 50% of the vital capacity may be used, with a consequent reduction in the reserve of respiration. In healthy individuals, the vital capacity is affected by the physical fitness, type of physique, occupation, sex, and age. It is slightly lower in females, and it decreases with advancing years. The absolute values for the vital capacity and the various subdivisions of the lung volume vary so widely that they are of little value in clinical application. However, if these are expressed as percentages of the total lung volume, the figures fluctuate within well-defined limits and are, therefore, of greater value.

Without determining the "residual air," however, it is impossible to compute the total lung volume. Unfortunately, no method has yet been evolved for determining the "residual air" which is practical enough for routine use at the bed-side. The principle which applies to all the various methods which are available at present is the one in which, if a known quantity of gas is distributed in an area of increased volume, the concentration of this gas will be lowered in proportion to this increase in volume. Hydrogen, oxygen, helium, or a mixture of these gases has been used. A known amount of the gas is mixed in the spirom while the patient breathes quietly. Sampl air are taken from the closed-circuit by mea the three-way cock at minute intervals analyzed for the concentrations of the diff gases. When these become constant, the res air is estimated. This is based on the fact tha nitrogen content of the alveolar air is consta 79%, the concentration of the test gas known, the total volume of the nitrogen co in the lungs can be determined. Because o impracticability of this method for routine cl use, it is not possible to estimate the total volume. With this, the estimation of the capacity as a percentage of the total lung vo cannot be determined.

A classification of vital capacity, how based on the sex and height of the individual been found to be sufficiently accurate for pra use. Healthy males have a vital capacity of or more, healthy females at least 85%, and a decrease occurs in elderly individuals. V which are below these figures, especially if are 65% or less, would indicate impairment alveolar function.

The total lung volume remains constant i same individual at rest. It is normally incr when greater demands are made on the respir as with physical exertion and oxygen-defici The residual air is also constant in the same vidual at rest, and the relation between the is fairly constant for healthy individuals o same sex. The residual air tends to increase advancing years, and if it is found to be more 35% of the total lung volume, impairmen alveolar function is present.

In the recumbent position, there is a redu in the total capacity, vital capacity and re air. Lying on one side results in a lower these values in the lower lung, whereas h values are found in the upper lung. The rel ship between the residual capacity and the capacity is a good indication of pulm efficiency. The higher the relationship, the marked does the dyspnea become.

There is for every individual in any given whether this is rest, exercise, or disease, a c ventilatory or "breathing requirement." At a normal individual may breathe 6 litres of one minute, whereas with exercise 25 litres may be breathed. If this extra ventilation ca place easily and without conscious effort, no toms will develop and no dyspnea will be pr But if this accommodation does not take easily and without effort, dyspnea will result

The "resting minute volume," which breathing requirement at rest with the p supine is estimated from the tracings of the respiration. For the breathing requirem exercise, a standard exercise test is used, con

of stepping up and down a 20 cm. step thirty times in one minute, and noting the volume of air that is breathed during the next five minutes. The use of walking as a test exercise more nearly approximates the patient's normal exercise, and can be used by any patient that is allowed to go to the bath-room. Instead of the spirometer, a one-hundred litre Douglas bag is used. This is completely emptied and attached to the patient by means of a mouth-piece with a one-way valve. This is carried by the examiner as the patient walks a standard distance of 720 feet in 4 minutes, the space and time being checked every 15 seconds. A hall-way measuring 180 feet to be covered in one minute will equally serve the purpose. As soon as the test is completed, the patient states whether he is not dyspneic; slightly dyspneic, laboured breathing being just noticeable; moderately dyspneic, in which the breathing is uncomfortable but he is able to continue; or severely dyspneic, being exhausted and unable to continue. The total expired air is expressed from the Douglas bag and measured by means of the gasometer. This gives the "walking ventilation" in litres per minute. The actual figures obtained vary from eight to thirty litres in different individuals, but the figures were constant for each patient.

The "maximum breathing capacity," or "maximum minute volume" is the maximum volume of air that can be ventilated by a patient in a unit of time, and is expressed in litres per minute. This was formerly determined by having the patient undergo strenuous exercise or by inhaling carbon dioxide, the ventilatory rate being measured when maximum hyperpnea had been reached. As these measures were often distressing to the patient, it was found that equally dependable figures were obtained with voluntary hyperpnea at the maximum rate possible. This test provides a measure of the actual maximum pulmonary ventilation capable by that individual. It is a more accurate measurement than the vital capacity which only measures a single breath without regard to time. The "maximum breathing capacity" not only measures the amplitude of breathing with the addition of the dimension of time, but it is more closely correlated to the physical limitations of the patient.

The test is performed by having the patient breathe into the closed-circuit of the spirometer, as hard and as fast as possible for thirty seconds. A satisfactory rate is about two breaths per second. At the end of 30 seconds, the total volume of air that has been breathed is measured and multiplied by two, corrections being made for temperature, barometric pressure and humidity. The normal value for men is 154 litres, for women 100 litres per minute. Any impairment of ventilatory function either by disease or injury is manifested by a lowering of the "maximum breathing capacity." If the figures are below 40 litres per minute in a male patient, or 35 litres in a female, the immediate post-operative course after a lobectomy or thoracoplasty may be stormy, and dyspnea on minimal exertion or even at rest may result.

"Breathing reserve" is the amount of ventilation which is still possible beyond the actual ventilatory requirement for a given state of activity. As the "maximum breathing capacity" is relatively fixed for that individual, the breathing reserve will vary in inverse proportion to the breathing requirement for the particular activity. This is usually expressed in terms of per cent of the "maximum breathing capacity."

There is a definite correlation between the "walking ventilation" (W.V.) and the "maximum breathing capacity," (M.B.C.). If the ratio M.V.: W.B.C. was less than 0.3, no dyspnea occurred. If the figures obtained were between 0.3 and 0.5, slight to moderate dyspnea occurred. If over 0.5, severe dyspnea developed.

The ratio of "breathing reserve" over "maximum breathing capacity" is also found to be of value in assessing pulmonary function. If the ratio is above 93%, no dyspnea develops. Varying degrees of dyspnea occur lasting up to 2 minutes in cases that show figures from 92 to 81%. Severe dyspnea for at least 2 minutes occurs in all cases with a ratio below 80%.

As implied above, the assessment of impairment in the respiratory aspect of pulmonary function is difficult owing to the complicated technique necessary for testing oxygen saturation of the arterial blood. By using the spirometer, however, an indication as to whether the arterial blood is sufficiently saturated with oxygen can be shown by demonstrating whether an "oxygen deficit" is present or not. In normal subjects at rest, the same amount of oxygen is absorbed from the inhaled air per minute, no matter whether the air is atmospheric, with 21% oxygen, or whether 100% oxygen is inhaled. In individuals who have decreased oxygen saturation of the arterial blood, inhalation of pure oxygen will result in a transitory increase in the absorption of oxygen into the body.

The spirometer is first filled with 400 cc. oxygen, the remainder being room air. A record of quiet breathing is taken with the patient in the recumbent position. The spirometer is then washed and filled with pure oxygen, and another record of quiet breathing is made. The absorption of oxygen is compared by measuring the slopes of both tracings. The anoxemic subject will show a relative increase in oxygen consumption during the first few minutes of the pure oxygen breathing, whereas a normal individual will show no alteration in the two tracings.

The alterations that may occur in the spirographic tracing in pulmonary disease are best demonstrated in a case of chronic obstructive emphysema. In this disease, no matter the degree of severity, the most constant finding is a retardation and prolongation of the expiratory phase of respiration. In mild cases this may be present only with deep breathing, but with advanced cases, this occurs with quiet breathing as well. On taking successive deep breaths, as for vital capacity, there is an inability for expiration to return to the previous expiratory level. This shows that with each succeeding breath, there is a corresponding increase in the reserve air. There is a decreased "maximum breathing capacity," the respirations being of small amplitude. By administering bronchodilator drugs, improvement in the above findings will occur.

Summary

Although the laboratory methods which are used in estimating pulmonary function have been described in considerable detail, it is important to emphasize that equally valuable information can be determined by clinical measures alone. The majority of the laboratory methods are still too elaborate for routine use at the bedside, and aside from a few specialized centres, most hospitals dealing with diseases of the chest are as yet not equipped for them.

A Sub-Committee on Pulmonary Function Tests (Ref. 8), in dealing with this problem, have recently suggested the following minimal requirements in the evaluation of a patient's pulmonary function:

1. Careful analysis of the history, with special enquiry as to the patient's reaction to daily activity and physical stress.

2. Physical examination, with special reference to the effect of mild exercise, especially noting the presence of an elevated pulse or respiratory rate and the use of the accessory muscles of respiration.

3. A fluoroscopic examination which attempts to evaluate the magnitude, speed, and distribution of pulmonary ventilation.

4. Inspiratory and expiratory chest x-rays for comparison of the relative position of the mediastinum and leaves of the diaphragm.

5. Determination of the maximum breathing capacity. This is the only laboratory method recommended to be of practical value for graphic recording and useful for following progress.

References

1. Birath, G., Lung Volume and Ventilation Efficiency, Acta Medica Scandinavica, Supplementum Clin. (154), P. A. Norstedt and Sœner, Stockholm, 1944.
2. Bloomer, W. E., Application of Tests of Respiratory Physiology for the Clinical Evaluation of Pulmonary Pathology, Yale Jour. Biology and Medicine, Vol. 20, No. 2, 1947, p. 135.
3. Cournand, A., Richards, D. W., Jr., Darling, R. C., Graphic Tracings of Respiration in Study of Pulmonary Disease, Am. Rev. Tuberc., Vol. 40, No. 5, 1939, p. 487.
4. Gilson, J. C., Hugh-Jones, P., Measurement of Total Lung Volume and Breathing Capacity, Clin. Science, Vol. 7, No. 3 and 4, 1949, p. 185.
5. Moncrieff, A., Tests for Respiratory Function, Medical Research Council, Spec. Report Series 198, 1934, H.M.S.O. London.
6. Warring, F. C., Jr., Ventilatory Function, Am. Rev. Tuberc., Vol. 51, No. 5, 1945, p. 432.
7. Warring, F. C., Jr., Simple Tests of Ventilatory Function for Use in the Sanatorium or Clinic, Am. Rev. Tuberc., Vol. 60, No. 2, 1949, p. 49.
8. Report of the Subcommittee on Pulmonary Function Tests, Am. Trudeau Soc., Am. Rev. Tuberc., Vol. 62, No. 4, 1950, p. 451.

UROLOGY

The Diagnosis of Ureteral Calculus

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Although ureteral calculus can be accurately determined in 98% of cases of patients suffering from this painful disease, diagnosis is not always simple. Its presence is being overlooked constantly, and in a series of cases studied recently, 35% had been previously operated upon for appendicitis. The attending physician is also likely to confuse ureteral stone with gall bladder disease, tabes dorsalis, duodenal ulcer, inflammatory processes of the female adnexa, and in rare instances with intestinal obstruction or pneumonia. It simulates diseases of the upper urinary tract, caused by other types of ureteral obstruction; such as ureteral stricture, ptosis of the kidney with kinking of the ureter, and extraneous pressure resulting from inflammatory lesions or neoplasms in the adjacent organs.

The symptoms of this condition are quite characteristic; they consist of pain in the form of renal colic, or continuous dull lumbar ache, and are usually accompanied by nausea, vomiting and gaseous distension of the abdomen, chills and fever, haematuria, frequency and dysuria. The previous passage of gravel or small stones should always lead one to suspect the presence of ureteral calculus.

The first step in ruling out appendicitis, gall bladder disease or other confusing abdominal lesions is the making of a careful urinalysis. A considerable number of patients present gross haematuria in whom the presence of blood in the urine is quite apparent. Careful microscopic examination of urine invariably shows erythrocytes which result from the scratching effect of the stone on the mucous membrane of the ureter. We have encountered microscopic blood in all of the cases of the ureteral stone that have come under our observation. Red blood cells may also be found

in the urine of the patient suffering from retrocaecal appendicitis, but in these no stone shadow is seen, the leucocyte count is higher, and the percentage of polynuclear-leucocytes is elevated. So with careful study, the differential diagnosis between ureteral stone and retrocaecal appendicitis can be accurately made. Albumin, leucocytes and micro-organisms are also found when concomitant infection is present. The presence of crystals is again of diagnostic value. These are usually of the variety which makes up the stone, and the finding of uric acid crystals in the patient suffering from uric acid stone which is non-opaque to x-ray, is of great diagnostic value.

In making the diagnosis of ureteral stone, the information obtained from investigation of the ureter by means of a cystoscope, passage of the ureteral catheter and the employment of the x-ray is invaluable. A certain percentage of stones situated in the lower portion of the ureter can be palpated (in the female) by digital exploration through the rectum or vagina. The cystoscopic picture is quite characteristic of stone. When the stone is situated in the intramural portion of the ureter it can sometimes be seen projecting from the orifice; in others the ureteral orifice is swollen, edematous and bulging. In patients in whom the stone is situated higher up in the ureter, the orifice usually appears congested. In introducing the ureteral bougie the sensation of resistance caused by the obstructing stone is of great diagnostic value. In passing the stone one often experiences a typical grating sensation. One may use a wax-tipped catheter or bougie in order to obtain the characteristic grooving, the significance of which was emphasized some fifty years ago. Great care must be adopted in passing these wax-tipped catheters or bougies through the cystoscope to prevent the wax being scratched while being passed through the cystoscope, thus producing erroneous conclusions. The diagnostic significance of resistance and gouging of the wax-tipped catheter is valueless in patients presenting bifid ureter, diverticulum or a greatly dilated ureter, because in these cases the catheter, in passing up the ureter, may fail to touch the stone. The employment of the x-ray enables one to clear the diagnosis in obscure suspected cases of ureteral stone. Because of the highly improved x-ray technique employed at the present time, very few stones are invisible to the x-ray, and even those that are slightly opaque can be visualized. The non-opaque, uric acid stone is rare, and is overlooked when one depends entirely on the x-ray for his diagnosis. The most valuable x-ray sign is the insertion of the opaque catheter or bougie into the ureter, and the demonstration of the contact of said catheter with the opaque stone. A calcified gland, phlebolith, sclerotic artery, enterolith, fecolith or foreign body in the intestines or shadow in the pelvic bones, may appear to be in contact with the opaque catheter and may be mistaken for ureteral stone. Stereoscopic films taken at different angles both in the lateral and anteroposterior positions, are necessary in order to clarify the diagnosis in these cases. On the other hand, the shadow cast by a stone located in the other limb of a bifid ureter, or in a diverticulum may appear to be out of the ureter. As duplication of the ureter occurs in about 4% of patients, further investigation by ureteral pyelography is necessary in order to detect the occasional ureteral calculus that lodges in the bifid ureter, or in a diverticulum of the ureter.

Retrograde pyelograms are of great assistance. They enable one to locate the stone in the ureter, to demonstrate its effect, and to visualize the amount of damage producing dilatation of the ureter and the pelvis above. The taking of fractional films after withdrawal of the catheter, or after the intravenous injection of one of the opaque solutions demonstrate the amount of obstruction caused by the stone. The employment of intravenous pyelograms is of great value, particularly in those cases in which one is unable to pass the ureteral catheter above the stone. Unfortunately, it is not as precise as retrograde pyelograms. When kidney function is inhibited, very little dye is excreted by the impaired organ, and poor uretrograms result. In fact, in some patients with impaired renal function we have noticed that the dye has not been eliminated in sufficient concentration to cast any shadow at all. In some of these cases suppression of renal function was not due to total destruction of the kidney, because, after the obstructing stone was removed, renal function returned to normal, as determined by making the indigocarmine and phenol-sulphonephthalein dye tests by subsequent employment of an intravenous pyelogram.

The diagnosis of ureteral calculus depends upon a careful history, complete urinalysis, employment of the x-ray and intelligent interpretation of findings obtained by cystoscopic study and ureteral catheterization. In differentiating ureteral calculus from appendicitis, gall bladder disease and other lesions of the gastro intestinal tract, the finding of blood in the urine is quite significant. Intravenous pyelograms indicate that the trouble is in the upper urinary tract. Precise diagnosis, however, still depends on cystoscopy, the passage of a ureteral catheter and the employment of the x-ray, including stereoscopic films and retrograde pyelograms. With intelligent interpretation of the findings sustained by the improved diagnostic methods now at our command, 98% of ureteral stones can be accurately diagnosed.

SURGERY

Arterial Lesions of the Extremities

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The study of trauma has become very important in modern life. Trauma to blood vessels is an important subdivision of the main subject.

It is rather difficult for a single individual to gain a large experience in blood vessel injuries in civilian practice. This fact is borne out by a statement made to me by Doctor Dan Elkin of Atlanta, noted vascular authority who told me that he sees about 18 aneurysm and arterio-venous fistulas per year in his civilian practice. What I shall endeavor to say will not be didactic or academic but will be information based on the work of others and on my experience in the laboratory, in the last war and in civilian practice. It will include failures and successes and will, I hope, be of some practical use to you as you encounter problems of vascular trauma in your own practice.

For general principles to be borne in mind when one is confronted with arterial insufficiency in an extremity: 1. Don't delay; 2. Don't heat; 3. Don't elevate; 4. Don't refrigerate.

I shall discuss all vascular injuries of the extremities with the exception of embolism which is more often due to heart disease than to arterial injury.

(A) Complete Severance.

This may result from a stab wound, a bullet, a piece of glass or sharp metal or the surgeon's knife.

In the case of smaller arteries ligation may readily be resorted to. In fact I believe that any artery in the upper extremity may be ligated with more or less impunity. Fine braided silk is the ligature material of choice and transfixion of the vessel is in order to prevent the possibility of slipping of the ligature. It is well to ligate the distal as well as the proximal cut end as reflux bleeding occurs from the distal end via the collateral circulation.

When a large artery such as the femoral above the profunda or the popliteal is completely severed it is well to consider some method of restoring the continuity. Retraction of the cut ends is usually so great as to preclude bringing them together for direct suture. The gap may be bridged by utilizing a vein graft. A section of the companion vein, great saphenous or external jugular may be used. The vein graft must be reversed when it is placed in the arterial system so that valves if present will not impede the flow of blood.

Slide 1 shows a vein graft which had been placed in the femoral artery of a human. It did not function because of intravascular clotting.

Slide 2 shows a vein graft which functioned satisfactorily for over two years between the severed ends of the left common iliac artery of a dog.

The vein wall has thickened and although it was dilated it presented none of the other features of an aneurysm. The vein may be sewn in place by the triangulation technic described by Guthrie in 1912 using 000 000 silk on an atraumatic needle or attached to the artery by means of Blakemore Lord vitallium cuffs[2]. Experience in either of these technics is best gained on the dog in the experimental laboratory.

Slide 3 shows a vein graft fastened in place in the left common iliac artery of a dog by means of Blakemore Lord cuffs. It functioned for 7 months but following exploration and examination of the graft it became filled with thrombus.

If the suture technic is used the patient should be heparinized for at least 72 hours. It is a difficult thing to keep the clotting time at 15 minutes. This is best accomplished by the continuous intravenous drip with 200 mg. (20 cc.) of heparin in 1000 cc. of saline or dextrose solution allowing 25 to 30 drops a minute. Each patient presents an individual problem and the number of drops per minute is regulated according to the clotting time. We are now experimenting with a pump which delivers a known quantity of fluid in a definite period. We hope with this method to eliminate the trouble we have had in maintaining a definite number of drops per minute.

Occasionally one may ligate a major vessel such as the popliteal artery and not lose the extremity. Such was the case illustrated in Slide 4. This slide shows the wound in the popliteal space of an air gunner whose popliteal artery was completely severed by a piece of "flak." The artery was ligated and the leg remained viable.

When it is found necessary to ligate a major artery such as the popliteal I believe it is wise to do a concomitant lumbar sympathectomy to insure maximum collateral flow.

(B) Laceration of an Artery.

This usually results from a knife, a piece of glass or sharp metal. If this condition is recognized early and repair of the laceration is effected the development of a false aneurysm is prevented. If the history is that of spurting bright red blood following a penetrating injury it is best to explore.

This slide shows a false aneurysm of an ulnar artery which could have been prevented by exploration and ligation at the time of injury.

American College of Surgeons, April 4, 1950, Winnipeg.

workman injured his fore-arm with a drill. Bleeding was stopped by pressure and the skin wound sutured. He later developed a pulsatile swelling and signs of ulnar nerve involvement.

When the laceration involves a major artery it is often possible to close the rent by vascular suture. Here again it is best to heparinize the patient for 72 hours if such a procedure can be adequately and safely carried out.

(C) Traumatic Vascular Spasm.

This may result from the impact of some substance against the artery or from stretching the artery. An example of the former would be a fragment of bone, an example of the latter would be stretching of the artery with a distracting apparatus in "setting" a fracture. Both of these causes have been observed.

If the signs of spasm i.e. a cold, pulseless, pale or mottled limb persist after the injection of papaverine, alcohol by mouth or intravenously, warming the trunk and contralateral limb and novocaine injection of the appropriate sympathetic ganglia then one must be prepared to explore the artery and consider arterectomy. As a matter of fact when exploration is carried out one may find something else upon which the above measures could have no effect. Here is a slide showing a foreign body which caused spasm of the popliteal artery.

Recently several authors, including Carter, Richards and Zachary[3], have drawn attention to the anterior tibial syndrome. This is a syndrome occurring in fit young men and consisting of ischemic necrosis of the muscles of the anterior tibial compartment of the leg with a lesion of the anterior tibial nerve. The pathogenesis is given as follows: unaccustomed exertion—muscle trauma —increased pressure within the anterior compartment—impaired blood supply to the affected muscles—ischemic necrosis. Although arterial spasm is not considered to be an important factor in the muscle necrosis it is convenient to consider it here as in our diagram of vascular injuries the artery is reduced in calibre, in this case by compression. It is believed that the condition can be prevented by graduated physical training.

If the syndrome is recognized early, rest alone may prevent irreversible damage to the muscles; but, if paralysis has developed, surgical decompression of the anterior tibial compartment as an emergency procedure is advocated.

(D) Local Obliteration (Thrombosis).

As mentioned previously on exploration of an artery you may find that you are not simply dealing with spasm. The artery may have been so disturbed that intima has been seriously damaged so that thrombosis has occurred thus blocking the flow of blood. This happens in the following case in which bird shot had contused the brachial artery but had not perforated it. Resection of the thrombosed section of the artery was carried out; note the distended distal portion of the artery showing that the collateral circulation was adequate. In the case of the femoral artery above the profunda or the popliteal artery one may have to consider a vein graft in addition to arterectomy.

Local obliteration of the brachial artery has been observed in a case of supracondylar feature of the humerus in a child. Exploration revealed that no amount of closed manipulation would have restored the radial pulse. Local resection of the damaged artery and vein graft, which I now don't believe is necessary in the upper extremity, resulted in restoration of the blood supply and Volkmann's ischemic contracture did not develop.

This shows a popliteal artery which was damaged by a high tibial fracture in which the lumen was obliterated by thrombosis. The case was seen late, gangrene developed and above knee amputation was necessary.

This slide shows a gangrenous foot which was seen three weeks after a dislocated knee had been reduced. The intima of the popliteal artery had been so damaged at the time of dislocation that local obliteration due to thrombosis occurred. It is well to be on the lookout for arterial insufficiency following the reduction of a dislocated knee.

(E) Secondary Hemorrhage.

This usually occurs as a result of sepsis. It is my impression that since the advent of chemotherapy and anti biotics the incidence of secondary hemorrhage has been reduced. Hemorrhage from the slipping of a ligature may usually be prevented by transfixing the vessel with a silk ligature as previously mentioned. When bleeding occurs the usual advice is to open the wound, identify the bleeding point, clamp it and tie it. In the presence of a necrotic mass in an infected wound this may not be easily done. In the extremities one may have to resort to ligature of the artery proximal to the wound. I know that this is a procedure which is not advocated but it was found necessary in this case. Following an above knee amputation this patient had 3 secondary hemorrhages. The last one could not be controlled by anything short of proximal ligature of the main artery. There was no deleterious effect on the skin flaps. Here, as in all cases, one hesitates to ligature the popliteal artery. When an artery is ligated it is well to remember not to do it in continuity—the artery must be severed.

(F) Delayed Traumatic Aneurysm and Arteriovenous Aneurysm.

Laceration of an artery which is not repaired may result in a traumatic aneurysm. A gun shot or stab wound, an auto or air plane accident or

some flying metal or glass may cause such a laceration[4].

Arterial aneurysms are more difficult to deal with than arteriovenous aneurysms or fistulas because the collateral circulation does not develop as well in the former as in the latter. Ligation of a minor artery and extirpation of the aneurysm may be dangerous. In suitable cases the continuity of the artery may be restored by a venous graft. Personally I prefer the suture technic rather than fixing the vein in the artery by means of Blakemore Lord cuffs. A strip of fascia may be placed around the vein graft in an effort to prevent too much distension of the graft. If the continuity is not restored all means at one's disposal should be used to improve the collateral circulation.

Arteriovenous Aneurysm or Fistula

In this discussion we are considering only the acquired type. Cases of acquired arteriovenous fistula seen in the past most often were caused by gun shot and stab wounds. With the increased incidence of serious automobile and airplane accidents cases of arteriovenous fistulas now arise from these sources[3].

Acquired arteriovenous fistulas are of two main types: the type in which the communication is direct between the artery and vein—and the one with an associated sac.

Collateral circulation develops well in arteriovenous fistulas. One is usually cautioned to wait 3 to 6 months before operating on arteriovenous fistulas to insure the development of maximum collateral circulation. I have operated as early as two weeks without any ill effects. There is a danger of waiting too long.

Here is illustrated a case seen three years after injury. This case was complicated by a false aneurysm superimposed on an arteriovenous fistula and required two operative procedures to correct the condition. The anatomy was so disturbed and the tissues so altered that we were unable to deal with the situation in one sitting. This is not the only case which I have seen a false aneurysm superimposed on an arteriovenous fistula due to rupture of the original sac or communication between the artery and vein.

The most practical method of dealing with an arteriovenous fistula in most cases is quadruple ligation—excision. Occasionally when the communication is a direct one the aperture in the artery may be repaired by a transvenous approach. The artery is repaired as one would repair an ordinary arterial laceration and the vein is sacrificed.

Arteriography is often useful in these cases in order to locate the site of the communication. One notes here where the vena comites suddenly became suddenly filled with the opaque media—this indicates the site of the communication.

I should like to say a few words about arteriography. We are using 35 per cent Diodrast. For an arteriogram on the lower extremity the patient is placed on a fracture table with the legs abducted. Two x-ray machines are placed so as to take an A.P. and lateral x-rays or to take two A.P. x-rays of different sections of the extremity. A skin test dose of Diodrast is placed on one of the fore-arms. If this is negative we proceed with the arteriogram. The patient is anaesthetized with Sodium Pentothal. The femoral vessels are exposed and a tape is placed around the artery and vein. With the circulation temporarily occluded by gentle traction on the tapes 40 to 50 cc's of 35 per cent Diodrast are injected into the artery. X-rays are taken as simultaneously as possible.

For the upper extremity the patient is placed supine on the operating table. The hand is suspended at right angles to the trunk. The brachial artery is exposed, the injection is made and the x-rays taken. An arteriogram often gives valuable information regarding the collateral circulation. A further use of the arteriogram is in differential diagnosis. In this case we thought we were dealing with an aneurysm of the lower femoral artery. The arteriogram showed the lower femoral artery to be normal with a large mass lying anterior to it.

Before closing I should like to mention a useful type of dressing for limbs which have been operated on for arterial injuries. It is a modification of the Sir Robert Jones pressure dressing. Its use here is not to apply pressure but to afford protection. I have observed a successful removal of a popliteal aneurysm complicated by osteomyelitis of the os calcis because the heel was allowed to rest on a splint for 5 days. A modified Robert Jones dressing would have prevented such a happening. It keeps the knee straight and offers protection by its bulk.

In Summary

I have tried to point out the important arterial injuries in the extremities as well as to mention certain points in their diagnosis and management.

It is to be hoped that lessons learned in this field may give us a lead in dealing more adequately with the so-called medical diseases of the vascular system of the extremities.

References

1. Guthrie, C. C.: Blood vessel surgery. Longman's, New York, 1912..
2. Blakemore, A. H. and Lord, J. W.: Blood vessel anastomosis by means of a non-suture method using Vitallium tubes. Chapter XII-A, New York, Thomas Nelson & Sons.
3. Carter, A. B., Richards, R. L. and Zachary, R. B.: The anterior tibial syndrome. The Lancet, Nov. 19, 1949 p. 928.
4. Pratt, S. H.: Surgical management of vascular diseases Lea & Febiger, Philadelphia, 1949.

CASE HISTORIES—SURGICAL

Scirrhous Carcinoma of Breast Radical Mastectomy

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This is the twelfth of a series of Case Histories which will appear in the Review each month. The purpose of these publications is not to present rare or unusual cases but rather to consider the routine management of common surgical cases.

Case No. A-5187. Miss D. W., St. Boniface Hospital. Color, white. Age, 37 years. Occupation, Comptometer Operator. Date of admission, May 13, 1948. Date of operation, May 14, 1948. Date of discharge, May 20. 1948.

Complaint on Admission

Lump in right breast, 4 years.

Present Illness

About 4 years ago (1944), following a blow on the right breast, patient noticed a small pea-sized nodule above the right nipple. This nodule was neither painful nor tender, nor did it increase in size during her menstrual periods. The nodule did not increase in size appreciably until about July, 1947, from which date it gradually grew and increased in hardness.

Inventory by Systems

Eyes—Vision good (wears corrective lenses). No diplopia or blurred vision.

Ears—Hearing good. No tinnitus or vertigo.

Respiratory—Infrequent colds and sore throats. No pains in chest, cough, expectoration, hemoptysis or shortness of breath.

Cardio-vascular—No history of rheumatic fever or syphilis. No chest pain, palpitation, dyspnoea or ankle oedema.

Gastro-intestinal—Appetite good. No food intolerance. No nausea or vomiting. No abdominal discomfort or pain. Bowels usually constipated. No diarrhoea or melaena. No history of jaundice.

Genito-urinary—No frequency or nocturia. No pain or burning on micturition. No hematuria.

Menstrual—Menarche, age 15 years. Periods always regular, interval 28 days. Duration 3 to 4 days. Amount of flow moderate. No dysmenorrhea. No intermenstrual bleeding or discharge.

Nervous system—No weakness or paralysis. No disturbance of gait. No sensory changes.

Musculo-skeletal—No pains or aches. Strength good.

Metabolic—No heat or cold intolerance. Loss of weight, 117 pounds to 108 pounds, 2 years.

Past History

Usual childhood illnesses. Gland removed from neck in childhood. Tonsillectomy and adenoidectomy, 1931. No accidents.

Mother—died at 78 years of age, old age.

Father—Died at 76 years of age, "hardening of arteries."

Two brothers—Alive and well.

Four sisters—Alive and well.

No history of tuberculosis, cancer, diabetes, epilepsy, mental disease, etc., in family.

Physical Examination

General Impression — A thin, pale-looking, white female, about 40 years of age, lying quietly in bed.

Head and Neck:

Cranial nerves—Intact.

Eyes—Lids, conjunctivae, corneae, pupils, lenses, show no significant abnormalities. Pupils equal in size, round, react briskly to light and accommodation. Ocular fundi show no pathology.

Ears—External ears, canals and tympanic membranes normal.

Nose—No obstruction. Mucosa clear.

Lips, teeth, gums and tongue—Normal.

Throat—Tonsils out. Mucosa shows slight congestion.

Neck—No cervical lymphadenopathy. Thyroid gland not visibly or palpably enlarged. Trachea in midline. Scar from lymph gland biopsy on right side of neck.

Chest:

Heart—Apex in 5th interspace 3½ inches from midline. Heart sounds of good quality, regular, 80 beats per minute. No murmurs. Blood pressure 120 mm. Hg. systolic, 80 mm. Hg. diastolic.

Lungs—No deformity of thoracic cage. Movements equal and symmetrical. Tactile fremitus good throughout. No dullness on percussion. Breath sounds normal. No adventitious sounds.

Mammae—The left breast is normal on inspection and palpation. Right breast, immediately above the right nipple and areola there is a firm, irregular, non-tender tumor approximately 1½ inches in diameter. This tumor moves freely over the underlying pectoral muscles, but is adherent to the overlying skin. The tumor does not transilluminate. On pinching the skin over the tumor, one produces a dimpling effect. There is no elevation or retraction of the right nipple. No discharge from the nipple. At the apex of the right axilla there is a firm, non-tender lymph gland, the size of a marble.

Abdomen—Scaphoid in appearance. No tenderness, guarding or rigidity. Liver and spleen not palpable. No masses felt. Intestinal sounds normal. Reflexes present and equal.

Rectal examination — Sphincter tone good. Uterus and adnexa show no pathologic change.

Back—Contour normal. No tenderness on percussion over vertebral column. Movement in all directions unrestricted.

Extremities:

Upper—No deformities or wasting. No limitation of movement. No clubbing of fingers.

Reflexes	Right	Left
Biceps	++	++
Triceps	++	++
Supinator	+	+

Lower—No deformities or wasting. No limitation of movement. No varicose veins. Dorsalis pedis and posterior tibial pulsations good. Vibration sense good.

Reflexes	Right	Left
Knee	++	++
Ankle	++	++
Babinski	V	V

Clinical Laboratory Findings

Urinalysis—Urine clear, straw-colored, neutral in reaction. Specific gravity, 1.013. Albumin, 0. Sugar, 0. Microscopic, 1-2 pus cells per h.p.f. No red cells. No casts.

Blood—Hemoglobin, 90%. Red blood cells, 4,450,000. White blood cells, 7,560. Differential leucocytes, polymorphonuclear, neutrophils, 67%, lymphocytes, 33%.

Wassermann—Negative.

Roentgenological reports—Chest, diaphragms are normal. Heart and great vessel shadows are normal. Lung fields are clear. Bony thorax is normal. Lumbo-dorsal spine, examination is negative.

Pre-operative Diagnosis

1. Carcinoma of breast.
2. Fibro-adenoma of breast.
3. Traumatic fat necrosis.

Indications for Operation

A firm, growing, non-painful, non-tender, solitary mass in a breast warrants exploration and biopsy.

Description of Operative Procedure and Operative Findings

Examination of both axillae, supraclavicular regions, opposite breast, pelvis, chest.

Position—Supine, with arm at right angles to the body and placed on an arm board. Painted with merthiolate entire arm, chest and upper abdomen. Hand and arm wrapped in sterile drapes.

Incision—was first outlined curving over the upper part of the arm, encircling the breast, planned so that it encircled the tumor with a wide margin of skin about 1 inch away. The part of the incision encircling the tumor was made and the entire tumor removed with a liberal margin of healthy tissue surrounding it. This was immediately handed to the pathologist. On cutting tumor across, hard grating, with yellowish streaks characteristic of malignancy. Rapid section, adenocarcinoma.

The wound was packed with gauze soaked in merthiolate and closed.

The upper part of the incision was now made and the skin flap dissected laterally as far as the insertion of the pectoralis major, removing subcutaneous tissue close to the skin. Then the medial skin flap was dissected just past the midline of the sternum. The insertion of the pectoralis major was now exposed by sharp dissection; the index finger of the left hand was hooked around the tendon and this was cut across close to its insertion into the humerus.

The muscle was then retracted medially to expose the coraco-clavicular fascia and pectoralis minor muscle. At this point the thoraco-acromial vessels were separated and ligated as they pierce the costo-coracoid membrane just above the upper border of the pectoralis minor.

The coraco-clavicular fascia was now incised to expose the pectoralis minor and this was severed at its insertion into the coracoid process. The long thoracic artery coursing along the lower border of the pectoralis minor was ligated. The pectoralis minor was pulled downward and medially. This entirely exposed the axilla. The axillary vein was found at its highest point, and the loose connective tissue, fat, and lymph glands were stripped away from the vein, by gauze dissection. Small vessels were ligated with chromic catgut 000. The fat and areolar tissue was then dissected off the posterior wall of the axilla, formed by the teres major, subscapularis, and latissimus dorsi. Here the subscapular vessels were cut and ligated; the long subscapular nerve was preserved. The serratus anterior was next cleaned off on the medial wall of the axilla, care being taken not to injure the long thoracic nerve. The entire axilla having been cleaned out, the entire mass of connective tissue, fat and lymph glands was displaced downwards and the fascia covering the latissimus dorsi, serratus anterior, and external oblique was incised along the posterior axillary line and dissected off cleanly by sharp dissection, working towards the midline. The lateral branches of the intercostal vessels were seized by forceps placed parallel with the chest wall, cut and ligated with chromic catgut 00.

The axilla was now examined again for any oozing and a hot, moist pack was placed in the axilla and dissected area while the rest of the operation was continued. The upper part of the rectus sheath was next incised and dissected upwards.

The entire mass of axillary glands, and pectoral muscles was pulled medially by an assistant and the mass was separated from the chest wall

by sharp dissection. The perforating branches of the internal mammary artery were isolated and clamped and ligated, and the entire mass was removed in one piece, leaving the axilla and entire chest bare.

All bleeders were tied with chromic catgut 000. The skin was sutured with fine black silk interrupted sutures. A small penrose was used for drainage. Gauze pads and dressings were applied with slight pressure by an elastoplast bandage to obliterate all dead spaces.

The arm was placed by the side and the forearm supported by a sling.

Anaesthetic

Pre-medication—Tuinal gr. iss. given the evening before operation. Morphine sulphate gr. 1/6 with atropine sulphate gr. 1/150 given I hour before operation.

Condition of patient—Temperature, 98.2°F. Pulse, 64. Respiration, 25. Blood pressure, 120/80.

Induction—With 2.5% sodium pentothal, 20 cc.

Maintenance—With nitrous oxide and oxygen.

Condition satisfactory throughout. 500 cc. whole blood and 500 cc. 5% dextrose in normal saline given intravenously during operative procedure.

Post-operative—Pulse, 72. Respiration, 15. Blood pressure, 100/70.

Gross and Microscopic Description of Tissues Removed

Gross—In upper outer quadrant of the right breast there is a very firm disc-like mass measuring 4 cm. in diameter and 2 cm. in thickness not adherent to skin or underlying muscle. Surface is somewhat nodular. In axillary fat only one node is grossly involved; it is firm, whitish and of bean size.

Micro—Adenocarcinoma, grade iii. Remaining breast shows some adenosis, but hyperplasia and cystic tendencies are not marked. Axillary node stuffed with neoplastic cells.

Final Diagnosis

Adenocarcinoma, grade iii, of right breast with metastasis to one axillary lymph node.

Progress Notes Including Post-operative Care During Stay in Hospital

May 15, 1948—Temperature, 100°F. Pulse, 80. Respiration, 20. Patient feeling well, spent a good post-operative night. Penrose drain shortened. Moderate amount of sanguinous discharge on dressing. Patient allowed out of bed. Fluid intake by mouth adequate.

Active and passive arm movements were begun on the second post-operative day.

The patient's temperature did not rise higher than 99.2° F. during the remainder of her hospital stay. The Penrose drain was shortened each day and removed completely on May 17, 1948. There were no post-operative complications and the patient was discharged, feeling well, on May 20, 1948, six days after her operation.

Condition on Discharge

Patient was in excellent condition on discharge; some limitation of movement at shoulder joint and guarding. Advised full use of arm.

Follow-up Notes Since Leaving Hospital

May 25, 1948—Seen at office. All sutures were removed. Small area (1 inch in diameter) of skin slough at axillary end of incision. Small collection of serum at upper end of incision evacuated. Patient has no complaints. Very little restriction of movement of right arm. No swelling or induration in right arm. Post-operative deep X-radiation was advised.

June 2, 1948—Adeno-carcinoma right breast. Post-operative x-ray treatment was directed to the axilla, right anterior chest wall and right supraclavicular region. Factors: anterior axilla and chest wall (15 x 15 cms.) and axillary (10 x 10 cms.) ports. 140 Kv.P. Target-skin distance; 50 cm. ¼ mm. Cu. and I mm. Al. filtration. Total dose to each area 1500 r (measured at skin distance). Posterior axilla and supraclavicular ports (10 x 10 cms.); 200 Kv.P. Target-skin distance 50 cm.; 2 mm. Cu. and 1 mm. Al. Total dose to each area: 1200 r (measured at skin surface). Irradiation administered over a period of three weeks. Two months after treatment, a second course of therapy will be given. (Dr. F. G. Stuart).

March 14, 1949—Patient examined. No evidence of recurrence found. Feeling very well.

July 19, 1950—Still no evidence of recurrence. No complaints. No limitation of arm movements or oedema of arm.

CARDIOLOGY

Advances in Coronary Disease

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Part 1

Part 2 to be published in the April issue

Atherosclerosis, and in particular coronary atherosclerosis, is by far the commonest of the fatal diseases of man in our Western civilization. Its leadership as the "captain of the men of death" is steadily increasing. Medicine and surgery are gaining control over many of the infectious, nutritional, and metabolic ills which have plagued mankind. An ever increasing portion of humanity is surviving these dangers to finally succumb to the neoplastic, degenerative, and other diseases before which we are still relatively helpless. This article will concern itself with a few of the newer concepts and developments in the battle against coronary disease.

The Problem of Atherosclerosis

"Man is as old as his arteries." This fatalistic concept implied that we were powerless to prevent or reverse the process of atherosclerosis because it was thought to be part of the natural process of aging. While great strides had been made in diagnosis and some advances in therapy of coronary disease, it is only recently that there has been much hope that we could discover the basic mechanism or find any specific therapy.

There have always been optimists who argued against the inevitability or aging concept of arterial disease. They pointed to pathological reports of aged people dying free from or with only minimal atheroma—and conversely to the many cases which are being reported of coronary disease in young people. The premature development of atherosclerosis in certain metabolic disorders such as diabetes indicated that something more than mere aging influenced the development of the disease. The reported low incidence in Oriental people further stimulated search for some factors apart from mere senescence which caused arterial disease to develop.

Intensive study has centred around the role of cholesterol in the aetiology of atherosclerosis since it was demonstrated 40 years ago that this compound was the largest constituent of atheromatous lesions. Diabetes and xanthomatosis are accompanied by high levels of cholesterol in the blood, and premature atherosclerosis occurs in these diseases. Myxedema and nephrosis also are accompanied by increased levels of cholesterol and it is stated by most authors that atherosclerosis develops in these conditions.

These facts led to animal experiments in the attempts to induce atherosclerosis artificially by cholesterol feeding. Anitschkoff first succeeded in producing hypercholestremia and atherosclerosis in rabbits by feeding cholesterol. There were many valid objections to drawing inferences from these experiments to the disease in man. The rabbit, being herbivorous, does not consume cholesterol under natural conditions. Therefore its metabolism is presumably not adapted to efficient disposal of this compound. Furthermore the blood level necessary to produce disease had to be very high, and the morphology of the lesions presented marked differences to that found in man.

In 1942, however, Dauber and Katz[1] succeeded in producing lesions in the chick—which does not use cholesterol normally in its diet—by feeding cholesterol and thiouracil (stilboestrol could be used instead of thiouracil). The lesions resembled those found in the human. In 1946 Steiner[2] successfully induced lesions in dogs. Of great importance is Stamler and Katz'[3] recent finding that they could induce atherosclerosis in the chick by feeding small amounts of cholesterol over a prolonged period and without raising significantly the cholesterol blood levels.

These successful animal experiments encouraged studies on the relationship of cholesterol and diet to atherosclerosis in man, but until recently these studies were unrewarding. Certain facts became established. Thus the level of cholesterol is shown to increase gradually with age and to level off in the decade 50 to 60[4]. The average concentration of cholesterol in known cases of coronary disease is higher than in the normal population. However that applies to the average only. There is a great overlap so that it cannot be predicted what the approximate level is likely to be in any individual case. No significant difference in the cholesterol content of the diet was found between groups manifesting a low or high cholesterol level in the blood[5]. Only on a very low cholesterol diet such as the rice diet was there any diminution in the cholesterol values, and even in these cases, as the diet was maintained over a period of months, the blood levels rose to their original figures. Gertler et al[6], who carried out these latter studies, concluded that fasting serum levels were uninfluenced by diet, but were maintained through endogenous synthesis of cholesterol.

Gertler also discussed the oft quoted evidence that Oriental peoples had a low incidence of atherosclerosis and hypertension. He pointed out that we really do not know the true incidence of these diseases in Oriental countries because of poor statistics and the high death rate from infections and nutritional diseases. In any case many factors such as heredity and environment, have to be considered before concluding that variations in diet

were the cause of any supposed differences in incidence of atherosclerosis.

This discrepancy between the definitive findings in animal experiments, and the failure in humans to establish an aetiologic relationship between exogenous cholesterol and atheroma led to various studies to determine if there was some ratio of cholesterol, cholesterol esters, phospholipids etcetera which would be revealing. Getler and co-workers[6] believed that there was a different distribution of these compounds in atherosclerosis and normal individuals. Others thought that the physical state of plasma lipids, or a protein combination with cholesterol might be of importance. Investigation of such complexes were not feasible by ordinary chemical methods, because it was necessary to break up the molecular groups as a preliminary step in cholesterol estimation. Physical, or non-chemical methods had to be devised.

A young medical physicist, Dr. John Golfman and his co-workers[7] at the Radiation Laboratories of the University of California have published studies which appear to be of fundamental importance. Their technique utilized the ultracentrifuge which was developed by Sverdberg, of Sweden, in 1940. This centrifuge had a speed and anti-gravity force many times that of conventional instruments. Speeds of about 50,000 revolutions per minute were used in Golfman's experiments. At this rate protein molecules will undergo sedimentation which can be measured in much the same way that red blood cells settle in an ordinary sedimentation tube under the influence of gravity. The heavier the protein, the more rapid was the sedimentation. It was thus possible to secure partitioning of the plasma into layers with proteins of different molecular weights in each layer. If the specific gravity of the solution was increased to exceed that of the proteins the same process occurred in reverse, that is the molecules underwent flotation rather than sedimentation. This was the method used by the investigators. The process of flotation, with the zones and boundaries produced by the different sizes of molecules were recorded by optical and photographic methods. About ten classes of lipoid bearing proteins were separated, and each one was assigned a name based on its rate of travel. The unit of measurement devised was the "Svendberg flotation (Sf)" unit equivalent to a speed of 1 x 10-13 cms., per second. Thus a 10 Sf protein is one that undergoes flotation at the rate of 10 x 10-13 cms. per second.

The lipoproteins were divided into four groups:
(1) Sf 75 and over (including the chylomicrons)
(2) Sf 30-70.
(3) Sf 10-20.
(4) Sf 3-6.

The two largest groups are increased after fat meals. They were not shown in this experiment to be related to atherosclerosis.

The two smaller groups do not change after a fat meal but are relatively constant. The smallest size, Sf 3-6, is not altered in atherosclerosis. . . . The group Sf 10-20 on the other hand seems definitely related in concentration to atherosclerosis. The molecular weight of these latter particles is about three million, formed of 70% protein and 30% cholesterol.

Presumably because its diet does not include cholesterol, the rabbits' serum does not contain the heavy molecules which are dependent on fat absorption, but does have two groups which are comparable to the Sf 3-6 and Sf 10-20 proteins in the human. In feeding experiments with cholesterol it was found that there was initial increase in the Sf 3-6 types and finally the Sf 10-20 group showed a higher level. It was only in those cases where the latter group increased that atherosclerosis developed, and the extent of the disease was related to the degree of increase in the serum levels of this Sf 10-20 compound. Measures such as the use of iodides which inhibited the formation of molecules of this group also prevented the development of atherosclerosis.

An investigation of 1,553 humans was undertaken. Two-thirds of these were controls of apparently healthy individuals. The rest included patients who had undergone an infarction, cases of angina without infarction, of hypertension, hypothyroidism, diabetes, and nephrosis.

In the control group below the age of 40, values of the Sf 10-20 molecules were low. In most cases they could not be measured (i.e., less than 5 mgms. per cent). After the age of 40 about half the normals had measurable values, the average normal being 15 mgms. per cent.

In the infarction group over 95% had measurable values, the average being 40 mgms. per cent. In angina pectoris, nephrosis, hypertension and hypothyroidism, the same results were found. Only a small number of diabetics were examined and the results were not as striking as in the other groups, except in young female diabetics, who had a high figure of the Sf 10-20 molecules.

The next step in the investigation was to determine whether the levels of Sf 10-20 molecules could be influenced by diet. This was found to be the case. Individuals manifesting high levels were placed on low cholesterol diets. Over a period of weeks marked reduction could be obtained in this cholesterol fraction.

Thus the authors believe they have proven that a special group of cholesterol-protein molecules are associated with atherosclerosis and that dietary measures can influence the level of these molecules.

They explain why there had never been good correlation between total cholesterol and atherosclerosis. While the total and Sf 10-20 cholesterol usually varied in the same direction, it was common for the values to be dissociated. It is possible

to predict that a patient with atherosclerosis will have an increased level of Sf 10-20 cholesterol molecules, whereas this could not be done in the case of total cholesterol. It is not possible to say, however, that finding a high Sf 10-20 figure indicates the patient has clinical atherosclerosis, because 50% of normal people over 40 had measurable values (although on the average these were much lower than in the pathological group). They make the obvious hypothesis that the controls showing increased levels either have subclinical atheroma, or are potentially so.

If the authors' findings are substantiated, and the theories based on them proven—a process which will take years—their work will indeed be a landmark in Medicine.

Another method of studying the lipids in the blood was described by Zinn and Griffith[8] in December, 1950. They examined the fat particles in the serum microscopically with a dark field, and made counts of these particles. They found that in the fasting state the chylomicrons, or large fat particles, were definitely higher in the sera of atherosclerotic and diabetic patients as opposed to normal controls. If correct it would indicate that the Sf 10-20 group of Golfman is not the only one affected in arteriosclerosis.

The Coronary Circulation

It was once taught that the coronary arteries were end arteries. This was true physiologically, because occlusion of a major coronary artery will always cause necrosis of the myocardium if the circulation was previously normal. There exist, however, numerous channels, which while inadequate in the face of a primary sudden stress, do dilate and increase under the stimulus of prolonged anoxia, and have a very important function in disease. Blumgart[9] showed that in the pig rich anastomotic channels developed in twelve days after partial coronary occlusion. These anastomoses protected the heart from subsequent complete closure of the vessel, no infarct developing. This sequence of events is known to have its counterpart in the human. These anastomotic channels also account for the occasional phenomenon of infarction at a distance, where infarction takes place not in the area supplied by an occluded coronary artery, but in an adjacent region which was being nourished by collaterals from the now obstructed vessel.

Infarction without occlusion of a coronary vessel may occur if there is a sudden drop in the volume of blood delivered to the entire coronary tree as a result of shock, haemorrhage, heart failure etcetera. This may cause subendocardial necrosis, to be described later.

Actual occlusion of a vessel is due in many cases primarily to subintimal haemorrhage rather than thrombosis. The haemorrhage causes obstruction by swelling of the vessel wall or by rupturing the arteriosclerotic plaque with subsequent thrombosis. Rare causes of occlusion are coronary embolism and syphilitic obstruction of the coronary ostia.

It is evident that infarction may occur without thrombosis, or even without coronary occlusion, and contrawise that occlusion need not cause infarction. Hence the term "coronary thrombosis" should be discarded as a clinical term in favour of "myocardial infarction."

Some years ago Blumgart and Schlesinger demonstrated that while in the majority of cases the posterior wall of the left ventricle and the septum derived their circulation both from both right and left coronary arteries, at times one or the other vascular supply predominated. In these cases they considered the circulation "unbalanced" since it threw a larger load on one vessel, occlusion of which resulted in a more extensive and unfavourable lesion than when both vessels shared in the blood supply.

It is thus interesting to speculate that anatomic differences in the coronary tree, plus the functional capacity to develop anastomosis, may determine to a large degree the prognosis in any case of coronary disease.

Clinical Syndromes

Myocardial infarction and angina pectoris are usually differentiated without difficulty. **Infarction** gives rise to severe prolonged pain coming on after effort or at rest, and unrelieved by nitroglycerin; or it may manifest itself with little pain but with severe shock or heart failure. The findings of fever, leucocytosis, increased sedimentation rate, together with distinctive and progressive electrocardiographic changes complete the diagnosis. Early ST segment alterations indicating acute myocardial damage, and Q waves caused by myocardial death, are necessary for the diagnosis. T wave changes without QRS alterations are indicative of ischaemia only; an intramural infarction may be present, but requires follow up studies and clinical correlation for diagnosis.

Angina pectoris typically is an acute attack of pain lasting less than 15 minutes, usually on effort, relieved by rest or nitroglycerin and apparently completely reversible without residual signs or symptoms. The electrocardiogram during an attack shows ST depression and T wave inversions. These indicate that the attack is due to a current of injury arising from deprivation of blood to the **subendocardium.** The usual explanations are as follows: (1) The coronary arteries supply first the outer layers of the heart, and the subendocardium is the last to receive its blood. (2) During systole the pressures in the heart muscle are highest bordering the heart chambers and will therefore offer the greatest impedence to blood entry in this phase.

Angina per se does not leave the heart unscarred. Pathologically it has been found that subjects who have had angina without known infarction universally show evidence of small infarcts or even occlusion of a major vessel. Some authors are of the opinion that the first attack of angina is always occasioned by a sudden major change in the coronary circulation due to marked narrowing or even occlusion of a large vessel. A sudden increase in severity of angina is due to a similar process.

A third syndrome intermediate between angina pectoris and myocardial infarction is becoming more defined as an entity. The patient has pain prolonged considerably beyond that of ordinary angina. The pain frequently comes on at rest and is not relieved by nitroglycerin. It thus suggests myocardial infarction. There are, however, no fever, leucocytosis, or sedimentation rate changes, and the electrocardiographic findings are evanescent. In other words there is no objective evidence of myocardial necrosis. The term coronary insufficiency is often used for this syndrome, but **"coronary failure"** has been suggested as a specific descriptive term. It is presumed that these attacks are due to a sudden change in the calibre of the coronary vessel or to acute diminution of blood supply from extra-coronary causes. In the former case they are often a warning of a further attack of true infarction. A period of bed rest and the use of anti-coagulants is therefore advisable after such an episode.

Subendocardial Infarction

Infarction resulting from a sudden diminution of blood to the entire coronary system, such as occurs in shock or hemorrhage is of special interest[12]. The infarction usually occurs in an area especially vulnerable because of a narrowed artery supplying such a region, producting the usual type of infarct. Sometimes, however, the anoxia affects the heart as a whole as it does in angina pectoris. The same region that is affected in angina will then suffer, that is the subendocardium. A rim of necrosis under the endocardium results. The electrocardiographic changes are the same as those occurring in angina pectoris—ST depressions—except that they are persistent rather than transient. The findings are the opposite of pericarditis, where subepicardial injury results in ST elevations.

Cardiac Hypertrophy in Coronary Disease

Myocardial hypertrophy is a common finding in coronary disease. Most authors believed that such a finding proved that hypertension was present or had existed sometime in the past. Friedberg[13] concluded that in the absence of hypertension hypertrophy might develop, but only if cardiac failure had been present.

A theoretical support for the idea that coronary disease per se can cause hypertrophy is suggested by the fact that hypertrophy is a feature of hearts in infants where the sole anomaly is an aberrant coronary artery. Failure, however, usually is present.

The most extensive pathological study of this problem which has yet been carried out necessitates a re-evaluation of our views on this subject. Yater et al[14] studied the pathological findings collected by the American Army Institute of Pathology of 450 young servicemen dying from myocardial infarction. Two-thirds showed hypertrophy of their hearts. In about 50% of the total the enlargement was of marked degree. Only 3% of the cases had hypertension during the last illness. Enlistment records and in most cases induction X-rays were available to indicate that hypertension could have been a cause of only a small number of cases of hypertrophy.

The large hearts tended to show more scarring than the smaller ones. Yater explains the cardiac hypertrophy on the sequence of, anoxia → reduction of muscle tone → stretching of muscle fibres → hypertrophy. The more frequent occurrence of old lesions in the large hearts would suggest that these hearts had been subjected to more prolonged and severe anoxia, thus resulting in hypertrophy.

Skeletal Lesions Following Myocardial Infarction

Pain and stiffness of the left shoulder is sometimes complained of following a myocardial infarct. This may be mild, or so severe as to constitute a "frozen shoulder." The lesion was presumed to be a periarthritis based on a mild pre-existing bursitis or other local condition. The pain of myocardial infarction, with resultant immobility and muscle spasm, brought to light and aggravated the local lesion.

Cases were described, however, where the condition progressed to involve the whole extremity, indistinguishable from causalgia due to local injury. There was pain, edema, cyanosis and eventually atrophy and trophic changes in the muscles and skin of the hand. The lesion was designated as the **"shoulder hand syndrome[15, 16, 17]."** The same changes have been described in other visceral diseases. The mechanism of this reflex dystrophy is explained as follows: An excessive stream of nerve stimuli arriving in the spinal cord from the diseased heart overflow into somatic motor neurons causing muscle spasm, and through sympathetic efferents causing vasospasm. The combination of muscle spasm and anoxia due to vasospasm produces the local lesions. These in turn now contribute disturbing impulses to the "internuncial pool" of neurons in the cord, thus creating a vicious circle and serving to perpetuate the local disease even after the original visceral cause is quiescent. Various forms of treatment are recommended for this dystrophy, in particular sympathetic block of the stellate ganglion.

Patients who are being treated for heart pain often come to the physician complaining of chest pains which are obviously not a part of the coronary disease but arise in the chest wall from the myofascial structures. No doubt "heart consciousness" makes these people apprehensive about minor chest pains which are usually ignored.

Rinzel and Travell[18], however, found that in patients with cardiac pain, local tender areas in various parts of the chest could usually be demonstrated. They reasoned that pain impulses were referred to the chest wall from the heart and created local areas which were no longer normal and which themselves now acted as a source of painful stimuli. These would reinforce subthreshold impulses from the heart to prolong the pain after infarction and increase the frequency and severity of angina.

They adopted a plan of treatment based on Wolff and Hardys[19] recommendation that, "When pain results from the persistence of primary visceral or other deep noxious stimulation and is associated with (somatic) hyperalgesia, its intensity may be modified by superficial and deep procaine infiltration in the hyperalgesic zones."

They proceeded to treat a group of patients with angina, and also patients with infarction who had prolonged pain, by infiltrating any local areas of tenderness they could find with procaine. They reported good results in pain due to infarction and in angina which followed infarction.

The reports of these authors offer an avenue of treatment which will be of practical importance, providing their results can be duplicated by other investigators.

(To be Continued Next Issue)

References

1. Dauber, D. V. and Katz, L. N.: Arch. Path., 34: 937, 1942.
2. Steiner, A. and Kendall, F. E.: Arch. Path., 42: 433, 1946.
3. Stamler, J. and Katz, L. N.: Circulation, 2: 705, 1950.
4. Gertler, M. M., Garn, S. M. and Bland, E. F.: Circulation, 2: 517, 1950.
5. Gertler, M. M., Garn, S. M. and White, P. D.: Circulation, 2: 696, 1950.
6. Gertler, M. M., Garn, S. M. and Lerman, J.: Circulation, 2: 205, 1950.
7. Golfman, J. W., Jones, H. B., Lindgren, F. T., Lyons, T. P., Elliott, H. A. and Strisower, B.: Circulation, 2: 161, 1950.
8. Zinn, W. J., Griffith, G. C.: Am. J. Med. Sc., 220: 597, 1950.
9. Blumgart, H. L., Zoll, P. M., Freedberg, A. S. and Gilliga, D. R.: Circulation, 1: 10, 1950.
10. Blumgart, H. L., Schlesinger, M. J. and Davis, D.: Am. Ht. J., 19: 1, 1940.
11. Freedberg, A. S., Blumgart, H. L., Zoll, P. M. and Schlesinger, M. J.: J.A.M.A., 138: 107, 1948.
12. Levine, H. D. and Ford, R. V.: Circulation, 1: 246, 1950.
13. Friedberg, C. K.: Diseases of the Heart, W. B. Saunders Co., Philadelphia, 1949.
14. Yater, W. M., Traum, A. H., Brown, W. G., Fitzgerald, R. P., Geisler, M. A. and Wilcox, B. B., Am. Ht. J., 36: 683, 1948.
15. Steinbrocker, O., Spitzer, N. and Friedman, H. H.: Ann. Int. Med., 29: 22, 1948.
16. Bayles, T. B., Judson, W. E. and Potter, T. A.: J.A.M.A., 144: 537, 1950.
17. MacLean, K. and Stranks, G.: Ca. Med. Ass. J., 63: 568, 1950.
18. Rinzler, S. H. and Travell, J.: Am. Ht. J., 35: 248, 1950.
19. Wolff, H. G. and Hardy, J. D.: Physiol. Rev., 27: 167, 1947.

ANAESTHESIOLOGY

Anaesthesiology Section

The monthly meeting of the Anaesthesiology Section of the Winnipeg Medical Society was held on Tuesday, February 6th, at 99 Niagara St. The scientific program was a Symposium on Spinal Anaesthesia, with Dr. D. C. Aikenhead as moderator.

Papers were read by Dr. Homer Eschoo on Physiology of Spinal Anaesthesia; Dr. David Tass, History of, Indications and Contra-indications for Spinal Anaesthesia; Dr. A. Natsuk, Techniques and Agents of Spinal Anaesthesia; Dr. Douglas Best, Complications of Spinal Anaesthesia.

Dr. F. A. B. Sheppard, as guest speaker, added to the Symposium with "Why a Surgeon Chooses Spinal Anaesthesia." Open discussion followed, and all present felt it was a most worthwhile evening. Exchange of views between members of the surgical team is an excellent thing, furthering knowledge in our common interest—The Patient.

Annual Meeting

The annual meeting of the Western Canadian Anaesthetists is being held in Calgary on February 21 to 24 this year. Guest speaker will be Dr. John J. Bonica, Director, Department of Anaesthesia, Tacoma General Hospital, Tacoma, Washington. There will be a full program of practical demonstrations and scientific papers. Dr. D. C. Aikenhead, of Winnipeg, will speak on "The Conduct of Anaesthesia for Removal of Rare Endrocrine Tumors."

Abstract

Preoperative Preparation and Choice of Anaesthetic Agents

Lloyd H. Mousel, Anaesthesiology, 11:495-500, July, 1950

Adequate preoperative preparation of surgical patients is one of the fundamental principles to be observed carefully if morbidity and mortality are to be kept to a minimum. Patients who are victims of cardiac or debilitating diseases deserve the benefit of special consideration and individual attention.

Cardiac Disease

Most patients with heart disease who can carry out nearly normal physical activity without dyspnoea or precordial pain and, who do not have pretibial edema or rales in the bases of the lungs are fairly good risks for anaesthesia and surgery. On the other hand, decompensated cardiacs must be prepared for operation with bed rest, digitilization and restriction of the sodium ion.

Overloading of the circulatory system with intravenous fluids during or after operation is hazardous. The patient with coronary or aortic heart disease must be protected from sudden lowering of blood pressure. Stormy inductions are to be avoided. Sodium pentothal to carry the patient into the first plane of surgical anaesthesia, will assure a quiet induction. Ether maintenance in a closed system, using a high concentration of oxygen, is almost always well tolerated by these patients.

Chronic Osteomyelitis

These patients are almost always in a state of nutritional deficiency with negative nitrogen balance. They are found to have decreased total blood volumes. Multiple transfusions given daily, or on alternate days, over a period of two weeks will usually correct the condition. These patients, with reduced total blood volumes, are actually in a state of chronic shock and will frequently react to spinal exactly as an acutely shocked patient responds.

Intestinal Obstruction

Patients with severe intestinal obstruction, who have vomited for a prolonged period of time, are also in a state of negative nitrogen balance with reduced total blood volumes and usually have a deficiency of chlorides. Inhalation anaesthesia is probably the safest method. Patients with acute intestinal obstruction without distention or vomiting may, under most circumstances, be operated on safely using the spinal technic.

Metabolic Diseases

Diabetes — The author does not believe that elective surgery should be done on diabetic patients until the blood sugar has been brought to a level 150 mg. per 100 cc. of blood, hydration is complete and there is no ketosis. If the diabetes is well controlled perhaps any anaesthetic method or agent might be used. The patient with severe uncontrollable diabetes or who comes in for emergency surgery may be anaesthetized with cyclopropane, spinal or with a combination of nitrous oxide or ethylene and sodium pentothal. Treatment of the diabetes should be instituted before or at the time of operation. Refrigeration anaesthesia for amputation of gangrenous lower extremities in severe diabetics is an acceptable and safe method.

Addison's disease and myasthenia gravis — These patients are poor risks. Spinals and curare are to be avoided.

Pancreatic diseases — Patients with malignancies or cysts can usually be handled with any anaesthetic agent or method. Patients with hemorrhagic pancreatitis, with extensive fat necrosis, are extremely poor risks and frequently die during or soon after operation, regardless of the type of anaesthetic employed.

Neoplastic Disease

Patients undergoing radical resection of the colon do better under a combination of spinal and pentothal anaesthesia with constant administration of oxygen. Patients with upper abdominal lesions, especially if the approach is transthoracic, usually do well under endotracheal ether-oxygen anaesthesia. Malignant lesions about the neck or throat can be resected under regional anaesthesia or with inhalation methods.

Asthma

It is important to plan carefully for the anaesthetic management of asthmatic patients. Excitement or worry may precipitate a severe attack during anaesthesia. Spinal anaesthesia is not satisfactory for most severe asthmatics. Acute attacks may occur during anaesthesia owing to the emotional state of the patient or to the paralyzing effect on the sympathetic nerves as the agent ascends in the middorsal region.

It has been the policy of the author to use small doses of avertin rectally in the patient's room. This drug, through sedative and basal anaesthetic actions largely eliminates the possibility of an asthmatic attack caused by emotional upset. It also tends to relax bronchial musculature. After the patient is in a state of basal narcosis he is taken to the operating room where gas-oxygen-ether, with ether maintenance is administered. Following operation, bronchoscopy is performed to remove as much foreign material as possible from the tracheo-bronchial tree.

The use of avertin to overcome bronchial constriction may be dangerous unless conservative doses are used. The average dose is between 40-60 mg. per kilogram body weight.

J. Phaneuf,
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St. Boniface Hospital.

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DERMATOLOGY

Skin Manifestations As An Aid In The Early Diagnosis of Internal Disease*

S. S. Berger, B.A., M.D.

Note: this paper was read on October 3rd, 1950, before the Annual Meeting of the Manitoba Medical Association. It was presented by means of some thirty printed charts and over seventy-five slides. Much of the interest of this paper is lost when transcribed to article form, because the paper was built around the slides. The charts which dealt chiefly with classification are included below with necessary explanations.

"The skin is a large organ—indeed the largest, with many complex structures and manifold diverse functions, e.g., horn formation, keratohyalin, basal cells, prickle cells, pigment formers, pigment storers, oxidation and reduction systems, enzyme systems, sweat glands, apocrine and sebaceous glands, at least eight different kinds of hair organs, nerves, several types of special sensory end-organs, glomus organs, blood vessels of many sorts and sizes, collagen and elastic structures, thermostatic regulatory apparatus, and equipment for lipoid, steroid and hormonal synthesis and for activation, storage and distribution of vitamins."

This complicated organ is exposed to many influences from within and to many forces from without.

The skin reacts to forces which attack it primarily, in a more or less independent and unique manner. The skin is also affected by internal and systemic influences and it reacts in relationship with other organs and with the body as a whole. It is this aspect which will be discussed below.

"The pathologic changes caused by the various exogenous and endogenous agencies in the highly differentiated cutaneous structures give rise to a great number and variety of distinctive patterns of disease."

Only those patterns on the skin which aid us in detecting internal disease will be discussed.

Many patients will not worry because of a sudden lack of the feeling of well being—or because of vague general complaints but they will become concerned and seek medical advice at the first appearance of a definite skin manifestation. The physician must always ask himself, "What now causes this patient to seek relief and what now brings this patient to me." This question can usually be answered by one or more of what might be called the following Four D's of Distress:

*Presented at the Annual Meeting of the Manitoba Medical Association, October 3rd, 1950.

The Four D's of Distress

Disfigurement—Acne Vulgaris.
Discomfort—Severe Pruritus.
Disability—Eczema of the hands.
Danger—Most patients with skin disease wish to know:

1. Is it contagious?
2. Does it leave permanent disfigurement?
3. Does it indicate some underlying serious derangement?
4. Is it due to
   (a) Getting the poison out of the system?
   (b) "Nerves," "Worries" or "Psychic Factors?"
   (c) Too rich blood?
   (d) Too much acid?

The disfigurement of Acne Vulgaris, the discomfort of severe itching and the disability of a severe eczema of the hands is reason enough to come to a physician. Often the correction of disfigurement, discomfort and disability will be a difficult task for general practitioners and specialists alike.

But for the patient who comes because of danger much can be done. "Many patients are filled with fear that their wart or mole is cancer, fear that their skin disease is highly contagious, will leave disfigurement, and that their skin eruption is a manifestation of some serious internal disease." The facts are that very few skin diseases indeed are easily transmissible, secondly that most skin diseases do not leave permanent disfigurement. The physician who can correctly and with assurance explain that the patient's skin disease is not cancerous, is not contagious, will not leave permanent scarring, is not due to internal disease, is not due to poison in the system, nerves, too rich blood, too much acid—will greatly relieve some of the patient's principal anxieties.

In addition to the above a knowledge of dermatology will help the physician to diagnose internal disease because of its early skin manifestations and it may enable the physician to check the disease. He may also be able to predict the possible future course and involvement of the disease because of its first appearance on the skin, and he may be able to institute prophylactic measures.

Indeed, Jonathan Hutchinson once said:

"The time is not far distant when diseases of the skin, instead of being esteemed as an unimportant if not repulsive specialty, will be regarded as offering unequalled opportunities for the study of morbid processes, and when they will take their proper place as introductory to the study of medicine, and, before trying to understand diseases which are to a large extent concealed from obser-

vation, the student will attempt first to master those which are exposed to view."

It is obvious one cannot discuss adequately all the early skin manifestations of the many internal diseases. It is hoped that the following presentation will illustrate to the physician how valuable a knowledge of dermatology can be to him.

First some easily recognized skin lesions which may be a part of early manifestations of many diseases will be discussed. Then will follow a review of some internal diseases that have early skin manifestations.

The plan of presentation is as follows: 1. Purpura, 2. Pigmentation, 3. Pruritus, 4. Erythema Multiforme, 5. Erythema Nodosum, 6. Fungous Diseases, 7. Vitamin Deficiencies, 8. Metabolic Disorders, 9. Endocrine Disorders, 10. Lupus Erythematous, 11. Sarcoidosis, 12. Nevoid Anomalies, 13. Neoplasms, 14. Blood and Blood Forming Organs, 15. Drug Eruptions, 16. Miscellaneous.

Purpura

This is a condition characterized by extravasations of blood in the skin and mucous membrane. The areas of discoloration may vary from pin point size to much larger areas known as ecchymoses. This is an easily recognizable skin manifestation and is often an important early manifestation of internal disease.

Classification of Purpura

A. Thrombocytopenic Purpura

Diminution in number of platelets.

1. Primary Thrombocytopenic Purpura (Essential, Werlhof's) (Impaired development of platelets).

2. Secondary Thrombocytopenic Purpura — secondary to (a) Blood Dyscrasias: Aplastic Anemia, Pernicious Anemia, Leukemia. (b) Infectious Diseases: Acute Disseminate Lupus Erythematosus, Sepsis e.g. Meningococcic, Typhoid Fever, Small Pox (Malignant type), Subacute Bacterial Endocarditis. (c) Drugs: Arsphenamines, Sulfonamides, Gold, Phenobarbital, Quinine, DDT. (d) Malignancy: Sarcomatosis, Carcinomatosis, Hodgkin's Disease.

Of principal interest to us as an aid in early diagnosis of internal disease is the Secondary Thrombocytopenic purpura group. Blood Dyscrasias, Infectious Disease, Drugs, and Malignancy, must always be considered.

B. Thrombasthenic Purpura

Qualitative Alteration in Platelets. 1. Familial, rare.

C. Purpura of Increased Capillary Fragility

Platelets normal.

1. Idiopathic, (a) Purpura Simplex, (b) Allergic Purpura, Anaphylactoid Purpura, Schonlein's triad, Purpura, Fever, Polyarthritis, Henoch's triad, Purpura, Abdominal Plain, Simulates Acute, abdomen, Fever.

2. Vitamin Deficiencies, Vit. C, Vit. K.

3. Toxic States, Drugs, Uremia (Uremic Products act as toxins), Endocrine Dysfunctions, Menopause, Menses, Diabetes.

Under purpura of increased capillary fragility of significant importance is the Allergic and Anaphylactoid Purpura. It must be thought of, if along with the purpura there is fever and addominal pain or polyarthritis. The purpura of increased capillary fragility is most commonly seen in dermatology. One must not forget that Vitamin Deficiencies and any toxic state can cause purpura.

Pigmentation

The physiology of pigmentation is not yet completely understood. There are many causes for pigmentation and it is difficult to classify them.

Pigmentary Disturbances of the Skin

I. Deposits of various heavy metals. (a) Argyria, (b) Arsenic Pigmentation, (c) Chrysiasis, (d) Bismuthia, (e) Tattooing, (f) Cold Cream Pigmentation.

Heavy metals administered by different routes and given medically may cause pigmentation. The pigmentation is from the metal itself and from increased melanin deposits which is secondary to the metal itself.

II. Increase in amount of Melanin Deposits. 1 Heavy Metals. 2. Chloasma, Pig. changes in localized or more extensive areas. (a) Idiopathic. (b) Secondary to Physical Agents, Chemical Agents. (c) **Symptomatic Chloasma:** Chloasma Uterinum, Lymphoblastomas, Tuberculosis, Hyperthyroid States, Hypothyroid States, Simmond's Disease, Cushing's Syndrome, Addison's Disease, Albright's Syndrome, Melano Epitheliomas, Chronic Alcoholism, Pellagra, Other Vitamin Deficiencies, Cachexia, Malaria, Tar Melanosis, Naevoid in Nature, In other Dermatoses.

The above is concerned with conditions that cause increased pigmentation due to melanin. Heavy metals we have discussed.

Chloasma refers to pigmentary changes in localized or more extensive areas, and it may be idiopathic, i.e., of unknown etiology or secondary to various physical causes such as heat, pressure, etc., and chemical causes such as toilet waters, external medicaments such as cantharides and mustard.

The Symptomatic Chloasma is most important for it includes many diseases as is shown above.

III. Other Pigments (a) Haemosiderin and Haemofucsin: Haemochromatosis (Bronze Diabetes), Trauma, Vascular Disturbances, Kaposi's Sarcoma, (b) Carotin, (c) Bile Pigmentation, (d) Unknown Pigmentation, Ochronosis.

The above represents a classification of Pigmentation due to pigments, other than melanin. Haemosiderin and Haemofucsin are derivatives of Haemoglobin.

Haemochromatosis is a disease of unknown etiology where in addition to blue gray pigmentation which is similar to Addison's Disease there is associated cirrhosis of the liver, sclerosis of the spleen and pancreas and usually associated Diabetes Mellitus.

The blue gray color of Haemochromatosis is to be distinguished from the pigmentation in Addison's disease, from the bluish red color seen in Polycythemia Vera and the discoloration of the skin seen in association with Sulfhaemoglobinemia and Methemoglobinemia. There is no pigmentation of the mucous membranes in Haemochromatosis as is seen in Addison's Disease.

Kaposi's Sarcoma: A rare disease entirely characterized by reddish brown or bluish red nodules in the skin. There may be metastases involving nearly every organ in the body. The disease may also start in the internal organs. Pigmentation is due to haemosiderin and haemofucsin deposits.

Carotin Pigmentation: Yellow discoloration of skin caused by certain vegetables and fruits consumed in excess quantities, e.g., carrots, oranges, cucumbers.

Bile Pigmentation as seen in Jaundice: Its importance as a manifestation of internal disease need not be stressed.

Ochronosis: The exact nature of this pigmentation is unknown. It is a rare disturbance characterized by pigmentation of the scleras and ears and dark color of urine and frequently associated arthritis.

Pruritis

I. Primary Essential or Idiopathic Pruritus.

II. Secondary Pruritus.

1. Physical Causes.

2. Chemical Causes. A. Exogenous Substances. B. Endogenous Substances. (a) Drugs, (b) Foods, (c) Inhaled substances, (d) Substances originating from foci of infection or infestation, (e) Normal Metabolites, (f) Abnormal Metabolites. 1. Autointoxication. 2. Thyrotoxicosis. (3) Menstruation. 4. Pregnancy. 5. Climactericum. 6. Carcinomas. 7. Blood Dyscracias. 8. Hodgkin's Disease. 9. Liver Diseases. 10. Kidney Diseases. 11. Pancreatic Diseases. 12. Diabetes.

III. Psychogenic and Neurogenic Pruritus.

A. Neurogenic and Organic. B. Neurogenic and Functional. C. Psychogenic and Organic. D. Psychogenic and Functional.

IV. Pruritus Accompanying Certain Dermatoses.

Pruritus is a very common skin manifestation. Primary, Essential or Idiopathic Pruritus: i.e., pruritus which seems to stand alone and which appears to be the sole manifestation or symptom and which is of unknown etiology does not concern us here.

Secondary Pruritus: is pruritus associated with definite pathologic dermatologic or other entities or caused by ascertainable external or internal factors.

Physical Causes such as heat and cold and pressure, or chemical causes due to exogenous substances such as stinging nettles, external medicaments, soaps, etc., also does not concern us. But endogenous chemical substances are important. Drugs of special importance are morphine and derivatives, quinine, sedatives, anti pyretics, laxatives. Foods, especially common sensitizers such as eggs, wheat, milk, fish, cheese. Inhaled substances, especially pollens, dust, silk, volatile substances in perfumes, etc. Substances originating from foci of infection or infestation, i.e., bacterial and fungous products and products of intestinal and other parasites, etc. Normal Metabolites of the body—either present in increased concentration or perhaps the patient has become hypersensitive to those present in normal concentration.

The section Abnormal Metabolites is very important for us for it embraces many internal diseases. It is felt that the reason for itching in most of these cases is due to the release of abnormal products of metabolism. Itching is often premonitory and the first sign of sickness in such patients.

Two examples will demonstrate this:

1. A woman of 45 with severe pruritus of the right side of the abdomen and right breast was found to have a tumor of the gall bladder.

2. A man, aged 40, had generalized pruritus discovered to be due thyrotoxicosis.

The possible connection between such general disorders and itching is important for two reasons.

1. Itching may precede all other symptoms, and search for underlying causes may therefore lead to early discovery of diabetes, cancer, blood dyscrasias, etc.

2. No causal treatment of the itching is possible unless the underlying abnormality can be identified and treated.

Psychogenic and Neurogenic Pruritus

(a) Neurogenic and organic—itching such as found in tabes, zoster, or when a tumor is present.

(b) Neurogenic and functional—itching associated with neuralgias.

(c) Psychogenic and organic as in tumors of the brain, brain infections, etc.

(d) Psychogenic and functional, i.e., itching due to disturbances of the psyche without known organic basis—as found under emotional influences such as fear, anger, surprise.

Pruritus Accompanying Certain Dermatoses

In some of the cases both itching and lesions are produced by underlying cause or causes.

Erythema Multiforme. This is an acute inflammatory disease characterized by crimson red and purplish red macules, papules, or nodules occasionally by vesicles or bullae and rarely by pustules. The lesions are variously grouped or isolated and prone to recurrence.

Below is an attempt to classify Erythema Multiforme.

1. Limited to the skin—Unknown etiology.

2. Symptomatic Erythema of known Infectious Diseases, e.g., Cholera, Glanders, Typhus, Measles, Septicemia, Malaria.

3. Surface Expression of Visceral Disease. (a) Gastro Intestinal Crisis, (b) Haematuria and Nephritis, (c) Cerebral Symptoms, (d) Pulmonary Complications, (e) Involvement of the Heart, (f) Infiltration of synovial sheaths, peri articular tissues and arthritis.

4. Cutaneous Manifestations Due to Drugs, e.g., Quinine, Arsenic, Belladonna, Chloral, Salicylic Acid, Iodides, Bromides, Penicillin, Sulfa Drugs.

Erythema Nodosum: Erythema Nodosum is a disease characterized by pea to fist sized nodules, pale red, pinkish to livid blue in color, and most commonly but not necessarily situated on the lower legs. There is no suppuration of the lesions.

Erythema Nodosum has no specific etiology. Below is listed most of the causes and what should go through one's mind after a case is recognized.

Erythema Nodosum

No Specific Etiology. 1. Any Infection May Cause it, e.g., Leprosy, Tuberculosis, Syphilis, Streptococcal Infections, Rheumatic Fever, Lympho Papio Venereum, Ulcus Vulvae Acutum, Ringworm of the scalp, Coccidioides Inmitis, Measles.

2. Other Causes: Temperature Changes, Malarial Chills, Dentition, Urethral Irritation, Drugs esp. Iodides and Bromides.

Fungous Diseases

1. Actino Mycosis: Cervico Facial, Thoracic, Abdominal.

2. North American Blastomycosis: Lung, Bone, Liver, Spleen, Kidney, Central Nervous System.

3. Moniliasis: Mouth, Endocardium, Vagina, Meninges, Bronchi, Lungs.

4. Histoplasmosis: Lungs, Spleen, Adrenal, Larynx.

Fungous diseases are often overlooked as a causal factor in internal disease. It is true they are not terribly common but they are nevertheless very important. Fungous diseases most often make their appearance on the skin. One should realize that the manifestation on the skin may indicate the presence of fungous disease in other organs, e.g., in Actinomycosis, or that there is the possibility that the cutaneous site is only the beginning of greater involvement.

The three types of actinomycosis are well known. They are the cervico facial, thoracic and abdominal. North American Blastomycosis—may be limited solely to the skin or involve many other organs. The same is true of moniliasis but systemic involvement is not too common. Note how widespread monilial infection may become.

Moniliasis of the vagina is common in diabetes and pregnancy. In diabetes the infection is related to the excess amount of sugar in the blood and urine, and in pregnancy to the excess of glycogen in the vaginal epithelium.

One more or less thinks of histoplasmosis as a diagnosis made by chest x-ray and skin tests. But ulcers of the tongue and lip may be the first lesions noted. Sometimes histoplasmosis begins as a small skin lesion and widespread ulcers of the skin may develop.

Vitamin Deficiencies

Vit. A: (1) Diseases Characterized by Hyperkeratinization, e.g., Phrynoderma or Toad Skin. (2) Sjorgen's Syndrome.

Vit. K: Purpura.

Vit. C: Scurvy, Petechiae, Ecchymoses.

Vit. B Complex: Vit. B1 (thiamine) Beri Beri; Vit. B2 (riboflavin) Perlèche, Cheilosis, Seborrhoea.

Vit. B1, Vit. B2, Nicotinic Acid: Pellagra.

Vit. B Complex: Acne Rosacea.

Vitamin Deficiencies

Vitamin A. One sees manifestations in the skin characterized by follicular hyperkeratosis—and dryness of the skin, for example: phrynoderma or toad skin.

Sjorgen's syndrome is characterized by marked dryness of the mucous membranes, especially the lacrimal and salivary glands and the mucous glands in the respiratory tract.

Vit. K deficiency results in Purpura which has been reviewed.

Vit. C deficiency results in scurvy. The most striking skin manifestation is purpura which has been discussed.

The Vitamin B Complex

Vit. B1—Edema of the skin and cyanosis and general itching, may be the first sign of beri beri.

Vit. B2 (Riboflavin)—Evidence of this deficiency is Perlèche—which is an inter trigo of the corners of the mouth.

Monilia has been mentioned as a causal factor in Perlèche. The question is not clear how or to what degree riboflavin deficiency affects the condition or whether it is entirely responsible for it, and the fungous is merely a secondary or superimposed agent.

Cheilosis—Macerated linear fissures at each angle of the mouth, with a red, scaly inflammation of the vermilion portion of the lips. One may see in a Vit. B2 deficiency a mild greasy seborrhoeic dermatitis on a slightly erythematous base in the naso labial folds.

Pellagra, of course, has symptoms arising from the skin, gastro intestinal tract, and nervous sys-

tem. Sharply demarcated symmetric lesions may appear on any portion of the body—but are most common over sites of irritation. Many lesions are restricted to exposed parts of the body and the dermal lesions follow exposure to sunlight. Glossitis and stomatitis are early and common symptoms.

Acne Rosacea is considered by some authorities to be due to a Vit. B complex deficiency. It is characterized by hypertrophy and telangiectasies, often associated with acneform lesions.

Metabolic Disorders

I. Disturbances in Carbohydrate Metabolism

A. Skin Disease in Frank Diabetes. 1. Dermatophytosis. 2. Recurrent Furunculosis. 3. Pruritus. 4. Moniliasis. 5. Erosea Interdigitalis Blastomycetica—an interdigital intertrigo usually seen in the third and fourth interspace. 6. Perlèche. 7. Paronychia of Monilia Type. 8. Necrobiosis Lipoidica Diabeticorum. 9. Xanthoma Eruptivum Diabeticorum.

B. Individuals with Latent Diabetes.

C. Dermatoses in Individuals with normal fasting blood sugar but high fasting skin sugar.

There are many skin diseases commonly seen in diabetes. A urine examination for sugar is part of every complete examination and urine should be checked for sugar when a patient complains of recurrent furunculosis, pruritus, moniliasis, etc. In some cases blood sugar may be indicated as in individuals with latent diabetes.

Necrobiosis Lipoidica Diabeticorum—is characterized by single or multiple yellow and red hard shiny plaques varying in size and occurring chiefly on the extremities. In 90% of the cases diabetes is associated and in 80% of the cases the patients are female. In 18% of a series of 80 cases described in the literature, cutaneous lesions appeared from 1-5 years before the symptoms of diabetes mellitus.

Xanthoma Eruptivum Diabeticorum is simply a sequel of diabetes in the course of which lipemia and hypercholesterolemia occur. We see Xanthoma tumors.

II. Amyloidosis

1. Skin involved only.

2. Systematized Amyloidosis. Skin, Gastro Intestinal Mucosa, Genito Urinary Mucosa, Musculature.

3. Generalized Amyloidosis of Internal Organs secondary to Syphilis, Tuberculosis, Chronic Suppurative Diseases.

Amyloidosis is protein in nature and appears to be a compound of albumin with chondroitin sulfuric acid.

There may be skin involvement only—or it may be involved with Gastro intestinal system, Genito urinary system and muscles. The skin is rarely involved in generalized amyloidosis.

III. Xanthomatosis

1. Xanthoma Tuberosum (Hypercholesterolemic Type). (a) Skin, (b) Xanthoma of Tendon and Tendon Sheaths, (c) Xanthelasma (Xanthoma of eye lids), (d) Xanthoma Plana (Palms and soles), (e) Xanthoma with Cardiovascular Involvement, (f) Xanthoma of Liver and Bile Ducts.

2. Xanthoma Disseminata (Normocholesterolemic Type). (a) Skin, (b) Osseous Xanthoma of skull, scalp, extremities and orbit, (c) Xanthomatous Involvement of Pituitary and Tuber Cenereum with Diabetes Insipidus.

3. Xanthelasma (Xanthoma of eye lids).

4. Secondary Xanthomatosis due to Lipemia.

5. Localized Xanthoma cell formation in true tumors.

Xanthomatosis is a metabolic disturbance also —occurring as a result of a disturbance of one or more of the body lipoids. The term Xanthoma appears to be a carry over from the observations of the dermatologist who first observed and reported this disease. Xanthoma means yellow tumor and is adequate to describe many of the skin lesions—but the term Xanthomatosis is not adequate to cover the variety of manifestations of the generalized disease which so frequently involves the internal organs and exhibits various cutaneous lesions, which do not necessarily appear as yellow tumors. Xanthomatosis has been classified as follows:

1. Xanthoma Tuberosum. Those with elevated cholesteral and blood lipoids. Note that there may be involvement of eyelids (Xanthelasma) tendon sheaths, palms and soles, liver, bile ducts, endocardium and blood vessels. The skin manifestations are yellow tumors on extensor surfaces chiefly. In 40% of 65 cases studied by Montgomery there was associated angina pectoris with Xanthomatous involvement of coronary arteries and occlusive vascular disease of extremities with resultant intermittent claudication. The hereditary factor is very important and we find the Syndrome of Cutaneous Xanthomata, hypercholesterolemia, and angina pectoris as a definite well defined entity in first, second, third or fourth generations.

2. Xanthoma Disseminata. Associated with normal cholesterol — shows fine papules and plaques predominating in flexor surfaces of the skin especially axillae, cubital and popliteal regions. Xanthelasma or involvement of eyelids may be seen in this group also. Note in this group there is possible involvement of skull, scalp and pituitary with associated Diabetes Insipidus.

3. Xanthelasma or Xanthoma of eye lids may occur in the elevated cholesterol type with skin manifestation on extensor surfaces or in the normal cholesterol type with skin manifestations on flexor surfaces, or the lesions may be limited to the eye lids alone. It may be a sign of early

cardiovascular disease, hypertension or arteriosclerosis. 70% of these cases can have elevated blood lipoids but not as marked as Xanthoma Tuberosum.

4. Secondary Xanthomatosis: due to lipemia—is seen for example in diabetes—as a sequel of the lipemia and hypercholesterolemia.

5. Localized Xanthoma Tumors—such as Naevo Xanthoendothelioma has no systemic involvement—blood lipoids are normal—lesions appear at birth and tend to involute in from 6 months to 3 years.

It is important therefore to differentiate Xanthoma Tuberosum from Xanthoma disseminatum because we can often predict the visceral organs involved by the type of skin lesions. In brief then Xanthoma Tuberosum has elevated blood cholesteral—has involvement of eyelids and extensor surfaces and has heredity manifestations. One must check for endocardial, vascular, hepatic and pancreatic disease. Xanthoma Disseminatum has normal blood cholesterol, involvement of eyelids and flexor surfaces—and has no history of familial incidence; and one must investigate bones, lungs, brain, and think of diabetes insipidus.

Endocrine Disorders

The Pituitary Body: Frohlich's Syndrome, Simmond's Disease, Acromegaly, Pituitary Dwarfism (Lorain-Levi), Cushing's Disease.

The Adrenal Gland: Addison's Disease, Adreno-Genital Syndrome, Cushing's Syndrome.

Skin manifestations may or may not help us in Endocrine disorders.

In Frohlich's Syndrome the skin is smooth and pale and well visualized. Cushing states that persistent freckles on exposed areas is characteristic of most cases of hypo pituitarism.

Simmond's Disease or Pituitary Cachexia: Early stage of the Syndrome resembles myxedema, the skin being waxy, pale and slightly edematous. Later the skin becomes dry, wrinkled and scaly or in some areas about the hands and feet, glossy, chill blain like or even sclerodermatic.

Acromegaly: The skin is overdeveloped and can be compared to a normal skin under a magniflying glass. The surface is coarse and the furrows deepened. Acne is not uncommon. Pigmentation and hair growth is increased.

Pituitary Dwarfism: Has dryness of skin, freckles and hypotrichosis of the body.

Cushing's Disease: Due to a basophil adenoma of the pituitary. Shows broad purplish striae atrophicae and the skin is often hyper pigmented. Hyper trichosis of the face and trunk has been constantly found in female and pre adolescent males. The picture of Cushing's disease may be a complication of cortisone and A.C.T.H. therapy.

The Adrenal Gland

Addison's Disease: Hyper pigmentation of the skin in connection with destructive adrenal disease is in the vast majority of cases an early symptom often appearing before asthenia and fatiguability. Pigmentation about the knuckles may be the first manifestation.

Adrenogenital Syndrome: Due to tumor of the adrenal cortex; Hyper trichosis is the outstanding skin manifestation in cortical tumors. It is often an early and conspicuous manifestation. It is, however, by no means diagnostic of adrenal cortical tumor. Unusual Acne and Comedones either in small children or in adults is almost invariably seen in cortical tumors. The Acne resemble the severe pyodermatic type known as Acne Conglobata.

Cushing's Syndrome: When a basophilic adenoma of the pituitary is absent and the clinical features of Cushing's Disease is present, the term Syndrome is used by George W. Thorn in Cecil's Test Book of Medicine. This syndrome is often seen in tumors of the Adrenal.

The Thyroid Gland

Hyperthyroidism: (1) Skin is smooth, thin, elastic, moist, warm, rosy; (2) Hyperhidrosis, (3) Overactivity of Sebaceous Glands, (4) Increased Pigmentation, (5) Vitiligo, (6) Edema, 7) Urticaria.

Hypothyroidism: (1) Skin is pale, often yellowish, cool, dry; (2) Edema, causing typical facies; (3) Hair Changes, (4) Nail Changes, (5) Low Resistance to Pyogenic infections.

The main skin manifestations of hyperthyroidism and hypothyroidism are listed above. The hair and nail changes in hypothyroidism are worth noting. The hair is dull, brittle, dry and easily wears off and sheds. The nails become brittle, decrease in size, have long and transverse grooving.

Myxedema:

A. Generalized Myxedema (with hypothyroidism).

B. Localized Myxedema (with hypothyroidism).

C. Localized Myxedema (without hypothyroidism).

D. Pretibial Myxedema.

Differences Between Two Main Types of Myxedema

Localized Myxedema with Hypothyroidism:	**Pretibial Myxedema:**
1. Morphology—Papular, Lichenoid, Nodular.	Plaque like swellings.
2. Distribution—Any part of body, rarely legs.	Pretibial areas.
3. Thyroid—With Hypothyroidism.	With past or present Thyrotoxicosis.
4. Thyroid Med'n—Response is good.	Response is nil.
5. Prognosis—Good with Thyroid Medication.	Spontaneous Remission after several years.

We are concerned with localized myxedema with hypothyroidism and Pretibial myxedema.

Pretibial Myxedema: As the chart shows occurs over the lower anterior aspect of the tibia. It is now thought to be caused by excessive production of thyroid stimulating hormone of the pituitary or the lack of its antagonist thyroxin. This condition usually occurs after operation for a toxic diffuse goiter with progressive exophthalmos. The reasoning being that either a large proportion of the thyroid cells has been surgically removed leaving no antagonist thyroxin, or the cells cannot rise to the stimulus and hence most of the thyroid stimulating hormone remains active.

Lupus Erythematosus

Chronic Discoid, Lupus Erythematosus.
Chronic Disseminate, Lupus Erythematosus.
Subacute Disseminate, Lupus Erythematosus.
Acute Disseminate, Lupus Erythematosus.

Lupus Erythematosus is classified into four types as shown above. The chronic localized discoid L.E., the chronic disseminate discoid L.E., the subacute disseminate L.E., and the acute disseminate L. E. The first two types are the chronic types. There are no systemic manifestations but the subacute and acute stages have. The subacute and acute stages may follow from the chronic types and do so in about one-third of the cases.

The systemic manifestations of Acute and Subacute L.E. are: Arthralgia, Malaise, Fever, Secondary Anemia, Aplastic Anemia, Leukopenia, Pernicious Anemia, Myeloid Immaturity, Increased Sed. Rate, Hemolytic Icterus, False Positive Wasserman, Reversal of A-G Ratio, Cardiac Murmurs, Bacterial Endocarditis, Renal Irritation, Splenitis, Hepatitis, Albuminuric Retinitis, Choroiditis.

Gastro intestinal disturbances associated with anorexia, nausea, vomiting, and diarrhoea, have led to erroneous diagnosis of cholecystitis, appendicitis and colitis.

Sarcoidosis: This is a granulomatous process related to tuberculosis. On the skin one sees nodules, papules and infiltrating plaques. Other organs where there may be involvement are:

(1) Bones, (2) Lymph nodes—tonsils, (3) Parotid and other salivary glands, Parotid and Iris—Uveoparotid fever, (4) Iris, (5) Iris and ciliary body—Iridocyclitis, (6) Spleen, (7) Liver, (8) Heart, (9) Suprarenal glands, (10) Other tissues.

Nevoid Anomalies

Von Recklinghausen's Disease: This is characterized by the presence of fibromata, café au lait spots and other nevoid growths. This disease is familial, inherited as a dominant genetic characteristic. The origin of the tumor is mesodermal from the connective tissues sheath of nerves. Usually there is a low standard of physical and mental development in the subjects of this disease. One must not forget that the neuro fibromata may involve the mucous membranes of the mouth, large nerve trunks, suprarenal capsule, intestines and bones.

Adrenoma Sebaceum: This disease is characterized by the occurrence usually on the face but occasionally on other parts of the body of small papules, telangiectasies, occasionally angiomata, associated with other nevoid conditions. Adenoma Sebaceum is seen in Bourneville's Disease or Tuberous Sclerosis of the brain, in which the brain cells are destroyed by neuroglial proliferation and potato-like tumors are formed. One may see epileptic seizures, mental deficiency, eye disorders, tumors and cysts in various organs, subungual fibrous nodules in gums, mouth and nose, and dystrophic changes in the nails.

Neoplasms

1. Paget's Disease of the Breast.
2. Sarcoma Cutis.
3. Kaposi's Sarcoma.
4. Malignant Melanoma.
5. Squamous Cell C.A.
6. Metastatic Carcinoma.
   (1) Direct Invasion from Subjacent Tumors.
   (2) Through the Lymphatics.
   (3) Through the Blood Stream.

Cutaneous Metastases:

Primary Carcinoma in Breast	50%
Stomach	15-31%
Lung	12%
Uterus and Kidney	9%

1. Paget's Disease of the Breast: The process begins with erythema and scaling—involving the nipple and areola. The surface becomes very red, often eczematoid, with weeping and oozing, like eczema, but unlike eczema the border remains sharply defined. One should look for an underlying carcinoma of the breast.

2. Sarcoma Cutis: The term designates solitary or multiple sarcomas of the skin. It therefore includes angiomatous, fibromatous, myxomatous, and lipomatous sarcomas. Broder found in studying 152 cases who had soft tissue sarcomas that in 62 cases the tumors arose from the subcutaneous fat and loose fibrous tissue and that in only 3 cases did it apparently originate in the deeper layers of the skin. It would appear therefore that many solitary and multiple sarcomas of the skin represent extensions from sarcomas of muscle, fascia, periarterial areolar tissue and nerves.

3. Kaposi's Sarcoma: This was mentioned previously under pigmentary disturbances where pigments deposited were haemosiderin and haemofucsin. This is a rare disease entity characterized by reddish brown or bluish red nodules in the skin. There may be metastases involving nearly every organ in the body. The disease may also start in the internal organs.

4. Malignant Melanoma: Usually begins as a blue-black nodule. Early in the course of the

disease metastases occur. It is the most malignant and rapidly fatal of all cutaneous neoplasms. Metastases may appear in the adjacent lymph nodes—but frequently these may escape and the first evidence of metastases may appear in any of the internal organs. The retina of the eye which contains pigmented cells is also a source of the neoplasm. Malignant melanoma arises in a mole. It is therefore important to remove all moles subject to continued irritation. It is important to remove moles on head and feet since these areas are the most frequent sites of the initial lesions of malignant melanoma. Any mole which exhibits growth or bleeds without sufficient provocation should be excised. The best time to do this is under the age of puberty.

5. Squamous Cell Carcinoma: Occurs not only in the skin but also in mucous membranes composed of squamous cells (buccal and genito-urinary orifices). In the skin it is rare except in the head and neck but it is prone to develop at the site of an old scar following a burn or extensive trauma. There are two forms, the papillary and the ulcerating. The ulcerating form is invasive, spreads rapidly and metastasizes to the neighboring lymph nodes early.

6. Metastatic C.A.—May reach the skin by 1. Direct invasion from subjacent tumors. 2. Though the lymphatics. 3. Through the blood stream. Of the cutaneous metastases, in 50% of the cases the primary carcinoma is in the breast, 15-31% in the stomach, 12% in the lung and 9% in the uterus and kidney. 33% of metastases of carcinoma of breast and 20% of other carcinomas have a predilection for the scalp.

Disorders of Blood and Blood Forming Organs

Lymphoblastomas: 1. Leukemia. 2. Lymphosarcoma. 3. Hodgkin's Disease. 4. Mycosis Fungoides.

A. Acute Leukemia: Petechial and Diffuse Haemorrhages, Ulcerations and Necrotic Lesions, Urticaria.

B. Aleukemic Leukemia: Haemorrhagic and other lesions.

C. Chronic Myelogenous: Skin manifestations are rare—are toxic, and non specific.

D. Chronic Monocytic: Macules and pale, shotty, papules simulating sec. syphilis, later slate blue in color. Purpuric, Haemorrhagic, Bullous Lesions. Eczematoid Plaques, Exfoliative Dermatitis.

E. Chronic Iymphatic Leukemia, Urticaria, Ecchymoses, Papular Lesions, Bullous Lesions, Exfoliative Dermatitis, Eczema, Herpes zoster (generalized), Hyper Pigmentation, Furunculosis, Impetigo, Alopecia, Tumors.

2. Lympho Sarcoma: (1) Primary Lympho sarcoma of the skin is rare. (2) Metastases of the skin from Lympho Sarcoma of other organs.

3. Hodgkin's Disease: Severe generalized non-specific pruritus, Generalized pigmentation, Urticaria, Nodules, Ulcers, Morbilliform Eruption, Scarlatiniform Exanthemas, Icterus, Alopecia, Recurrent Haemorrhages, Herpes Zoster, Exfoliative Dermatitis.

4. Mycosis Fungoides: Skin usually, other organs rarely.

The lymphoblastoma group is a very important group. When one sees any unusual and puzzling eruption on the skin one should always think of this group—and also of a drug eruption. Skin manifestations may be early signs of any of the lymphoblastomas.

Blood and Blood Forming Organs Continued

Anemia (no attempt at classification): Pallor is seen in practically all anemias.

Pernicious Anemia: Pallor, Yellowish discoloration, Pigmentation, Glossitis.

Hypochromic Anemia: Cheilitis, Perlèche, Buccal leukoplakia, Tongue changes, spoon nails.

Granulopenia: Buccopharyngeal lesions, Cyanosis of the face, Cutaneous ulcerations about body openings, Erythematous, pemphigoid, vesicular, papular eruptions.

Polycythemia Vera: Redness of skin and conjunctiva, telangiectasies and petechiae, rosacea.

Lesions in Drug Eruptions

Any form is possible from Simple Erythema to Gangrene.

1. Eczematous Eruptions (Erythema, Vesicles, Weeping), Quinine, ephedrine, mercurials, arsphenamine, sulfa group, penicillin, formalin, atabrin, procaine and other local anaesthetics.

2. Urticaria and Angioneurotic Edema: Belladonna, atropin, morphine group, phenolphthalein, sulfa group, penicillin, barbiturates, salicylates, iodides, bromides, arsenicals, amphetamine sulfate.

3. Scaly, Erythematous (Sarletiniform Morbilliform, and Dermatitis Exfoliativa—like: Arsenic, arsphenamine, belladonna, balsams, heavy metals, sulfa group, penicillin.

4. Erythema Multiforme — like: Salicylates, phenolphthalein, antipyrin, barbiturates, antibiotics.

5. Erythema Nodosum—like: Iodides, bromides, salicylates, sulfonamides.

6. Acneform Furunculoid and Erysipelas—like: Bromides, iodides, chlorides, oils and tars.

7. Pemphigoid and Erysipelas—like: Bromides and iodides, sulfonamides.

8. Purpuric: Iodides, arsphenamines, particularly sulpharsphenamine, balsams, Carbamides (sedormid), barbiturates, sulfonamides, D.D.T.

9. Lichenoid and Lichen Planus—like: Arsenic, arsphenamines, atabrin and other acridin and acriflavin derivatives, gold, amphetamine sulfate.

10. Fixed Eruption: Phenolphthalein, antipyrin, antipyrin, arsphenamines, barbiturates, sulfa

arsphenamines, barbiturates, sulfa group, quinine, salicylates, phenacetin, atabrin, gold and others.

Drugs may be responsible for practically any skin manifestation ever seen. Any form is possible from simple erythema to gangrene. One should always think of it when one sees any puzzling skin eruption. Above are listed the common lesions in drug eruptions—with some of the drugs most likely to cause the various eruptions.

Miscellaneous

Syphilis, Communicable Diseases, Tuberculosis, Chronic Ulcerative Colitis.

Omitted are the common skin manifestations of syphilis and communicable diseases. These are well known and recognized and one need only mention them in passing. Tuberculosis of the skin is rare in this country.

Chronic Ulcerative Colitis in a small percentage of cases is complicated by skin disease. The ulcerations which may occur on the lower legs are most troublesome and conspicuous. They increase rapidly in size often taking on a kidney shape. They are painful and quite deep—but the healing tendency is marked. The appearance and disappearance of the skin lesions may reflect the course but not the extent of the colitis.

References

1. Skin Manifestations of Internal Disorders: Weiner.
2. Diseases of the Skin: Ormsby and Montgomery.
3. Text Book of Medicine: Cecil.
4. General Remarks on the Management of Skin Diseases with Special Reference to Acne Vulgaris: M. B. Sulzberger and S. Berger. King's County Medical Bulletin, March, 1949.
5. Pruritis and Its Treatment: M. B. Sulzberger and J. Wolf. Medical Clinics of North America, November, 1935.
6. Dermatology and Its Relation to General Medicine: M. B. Sulzberger. New York State Journal of Medicine, September 15, 1932.
7. Xanthomatosis: Lawrence K. Halpern, M.D. Barnard Free Skin and Cancer Hospital.
8. The Mucinoses. A Classification with Histochemical Studies on the Nature of Mucin: Lawrence L. Palitz and Matthew J. Brunner. Journal of Investigative Dermatology, March, 1950.
9. The Association of Progressive (Malignant Exophthalmos and Localized Myxedema: Arthur C. Curtis, Edward P. Cawley and Edgar B. Johnwick, M.D. Archives of Dermatology and Syphilology, September, 1949.
10. Exophthalmos and Localized Myxedema: J.A.M.A., Dec. 10, 1949.
11. Manual of Medical Mycology.

BOOK REVIEW

Books on the Eye for Practitioners

Encyclopedia of the Eye Diagnosis and Treatment, is described in the preface as a text that has been written for those who desire a ready reference to the diagnosis and treatment of the more common ophthalmologic problems.

Ocular diseases, abnormalities and symptoms are set forth alphabetically and additional guidance is given by a comprehensive index. The amount of space given to each topic depends upon its importance but emphasis is laid on diagnosis and treatment. General practitioners especially should find the book a valuable aid.

Encyclopedia of the Eye Diagnosis and Treatment by Conrad Berens, M.D., F.A.C.S. Executive Eye Surgeon; New York Eye and Ear Infirmary; Professor of Clinical Ophthalmology, Post-Graduate Medical School, New York University; President, Pan-American Association of Ophthalmology; Managing Director of Ophthalmological Foundation, Inc.; President, Snyder Ophthalmic Foundation; and Edward Siegel, M.A., M.D. Attending Ophthalmologist, Champlain Valley Hospital, Plattsburg, New York; Associate Ophthalmologist, Physicians Hospital, Plattsburg. 76 Illustrations, including 42 subjects in color; 272 pages: J. P. Lippincott, Montreal. Price $5.75.

Essentials of Ophthalmology is the latest addition to the popular "Essentials" series. The author writes, "The material is presented in such a way that the physician in general parctice may use it as a handbook of essential information on the eye and its diseases."

In order to give a solid foundation, chapters are devoted to anatomy, physiology and examination. There is also one on the theory and practice of spectacle-fitting.

The second part of the book deals with diseases of the eye. These are arranged according to the principle part affected. The causes, diagnosis and treatment are given for each disease and stress is laid upon what the general practitioner can do in his office. The book is well illustrated.

Essentials of Ophthalmology, by Roland I. Pritikin, M.D., F.A.C.S., F.I.C.S. Eye Surgeon Rockford Memorial, Winnebago County and Swedish-American Hospitals, Consulting Ophthalmologist, St. Anthony Hospital, Rockford, Ill. 215 Illustrations, including 18 subjects in color; 561 pages: J. B. Lippincott, Montreal. Price $8.00.

Medico-Literary

J. C. Hossack, M.D., C.M. (Man.)

Oriental Practice

When the mob has raised you to fame, patients of a better class will slowly appear on the scene. After some coquetting about "etiquette," whether you are to visit them, or they are to call upon you, they make up their minds to see you, and to judge with their eyes whether you are to be trusted or not; whilst you, on your side, set out with determination that they shall at once cross the Rubicon —in less classical phrase, swallow your drug. If you visit the house, you insist upon the patient's servant attending you; he must also provide and pay an ass for your conveyance, no matter if it be only to the other side of the street. Your confidential man accompanies you, primed for replies to the "fifty searching questions" of the "servants' hall." You are lifted off the saddle tenderly, as nurses dismount their charges, when you arrive at the gate; and you waddle upstairs with dignity. Arrived at the sick room, you salute those present with a general Peace be upon you," to which they respond, "And upon thee be the peace and the mercy of Allah, and his blessing." To the invalid you say, "There is nothing the matter, please Allah, except the health"; to which the proper answer—for here every sign of ceremony has its countersign —is, "May Allah give thee health." Then you sit down, and acknowledge the presence of the company by raising your right hand to your lips and forehead bowing the while circularly; each individual returns the civility by a similar gesture. Then inquiry about the state of your health ensues. Then you are asked what refreshments you will take: you studiously mention something not likely to be in the house, but at last you rough it with a pipe and a cup of coffee. Then you proceed to the patient, who extends his wrist, and asks you what his complaint is. Then you examine his tongue, you feel his pulse, you look learned, and —he is talking all the time—after hearing a detailed list of all his ailments, you gravely discover them, taking for the same as much praise to yourself as does the practising phrenologist for a similar simple exercise of the reasoning faculties. The disease, to be respectable, must invariably be connected with one of the four temperaments, or the four elements, or the "humours of Hippocrates." Cure is easy, but it will take time, and you, the doctor, require attention; any little rudeness it is your power to punish by an alteration in the pill, or the powder, and, so unknown is professional honour, that none will brave your displeasure. If you would pass for a native practitioner, you must finally proceed to the most uncomfortable part of your visit, bargaining for fees. Nothing more effectually arouses suspicion than disinterestedness in a doctor. I once cured a rich Hazramaut merchant of rheumatism, and neglected to make him pay for treatment; he carried off one of my coffee cups, and was unceasingly wondering where I came from. So I made him produce five piastres, a shilling, which he threw upon the carpet, cursing Indian avarice. "You will bring on another illness," said my friend, the Haji, when he heard of it. Properly speaking, the fee for a visit to a respectable man in 20 piastres, but with the rich patient you begin by making a bargain. He complains, for instance, of dysentery and sciatica. You demand £10 for the dysentery, and £20 for the sciatica. But you will rarely get it. The Eastern pays a doctor's bill as an Oirishman does his "rint," making a grievance of it. Your patient will show indisputable signs of convalescence: he will laugh and jest half the day; but the moment you appear, groans and a lengthened visage, and pretended complaints, welcome you. Then your way is to throw out some such hint as

"The world is a carcass, and they who seek it are dogs."

And you refuse to treat the second disorder, which conduct may bring the refractory one to his senses. "Dat Galenus opes," however, is a Western apothegm: the utmost "Jalinus" can do for you here is to provide you with the necessaries and comforts of life. Whatever you prescribe must be solid and material, and if you accompany it with something painful, such as rubbing to scarification with a horse-brush, so much the better. Easterns, like our peasants in Europe, wish the doctor to "give them value of their money." Besides which, rough measures act beneficially upon their imagination. So the Hakim of the King of Persia cured fevers by the bastinado; patients are beneficially baked in a bread-oven at Baghdad; and an Egyptian at Alexandria, whose quartan resisted the strongest appliances of European physic, was effectually healed by the actual cautery, which a certain Arab Shaykh applied to the crown of his head. When you administer with your own hand the remedy—half a dozen huge bread pills, dipped in a solution of aloes or cinnamon water, flavoured with assafoetida, which in the case of the dyspeptic rich often suffice, if they will but diet themselves —you are careful to say, "In the name of Allah, the Compassionate, the Merciful." And after the patient has been dosed, "Praise be to Allah, the Curer, the Healer"; you then call for pen, ink, and paper, and write some such prescription as this:

"In the name of Allah, the Compassionate, the Merciful, and blessings and peace be upon our

Lord the Apostle, and his family, and his companions one and all. But afterwards let him take bees-honey and cinnamon and album graecum, of each half a part, and of ginger a whole part, which let him pound and mix with the honey, and form boluses, each bolus the weight of a Miskal, and of it let him use every day a Miskal on the saliva, i.e., on an empty stomach. Verily its effects are wonderful. And let him abstain from flesh, fish, vegetables, sweetmeats, flatulent food, acids of all descriptions, as well as the major ablution, and live in perfect quiet. So shall he be cured by the help of the King, i.e., the Almighty, the Healer. And the Peace (i.e., adieu)."

The diet, I need scarcely say, should be rigorous; nothing has tended more to bring the European system of medicine into contempt among Orientals than our inattention to this branch of the therapeutic art. When an Hindi or a Hindu "takes medicine," he prepares himself for it by diet and rest two or three days before adhibition, and as gradually, after the dose, he relapses into his usual habits; if he break through the regime it is concluded that fatal results must ensue. The ancient Egyptians, we learn from Herodotus, devoted a certain number of days in each month to the use of alteratives, and the period was consecutive doubtless in order to graduate the strength of the medicine. The Persians, when under salivation, shut themselves up in a warm room, never undress, and so carefully guard against cold that they even drink tepid water. When the Afghan princes find it necessary to employ Chob-Chini, (the jin-seng, or China root, so celebrated as a purifier, tonic, and aphrodisiac) they choose the spring season; they remove to a garden, where flowers and trees and bubbling streams soothe their senses; they carefully avoid fatigue and trouble of all kinds, and will not even hear a letter read, lest it should contain bad news.

When the prescription is written out, you affix an impression of your ring seal to the beginning and to the end of it, that no one may be able to add to or take from its contents. And when you send medicine to a patient of rank, who is sure to have enemies, you adopt some similar precaution against the box or the bottle being opened. One of the Pashas whom I attended—a brave soldier who had been a favorite with Mohammed Ali, and therefore was degraded by his successor—kept an impression of my ring in wax, to compare with that upon the phials. Men have not forgotten how frequently, in former times, those who became obnoxious to the State were seized with sudden and fatal cramps in the stomach. In the case of the doctor it is common prudence to adopt these precautions, as all evil consequences would be charged upon him, and he would be exposed to the family's revenge.

Burton, "Pilgrimage to Al Medina."

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University of Manitoba, Faculty of Medicine

REFRESHER COURSE PROGRAM

Arranged by the Committee on Post Graduate Studies

Winnipeg, March 26th, 27th, 28th, 29th, 30th 1951

Guest Speakers

Dr. Gaylord W. Anderson
Mayo Professor and Director of School of Public Health, University of Minnesota.
President-Elect, American Public Health Association.

Dr. Douglas E. Cannell
Professor of Obstetrics and Gynaecology, University of Toronto.

Dr. Ray F. Farquharson
Professor of Medicine, University of Toronto.

Dr. Walter C. MacKenzie
Professor of Surgery, University of Alberta.

Monday, March 26th

Morning

Health Officers' Association Program.

Registration for Refresher Course at Fort Garry Hotel.

Noon

12.30 **Luncheon — Fort Garry Hotel.**
Chairman—Dean L. G. Bell.
Guests—
Hon. Ivan Schultz, Minister of Health and Public Welfare.
President Gillson, University of Manitoba.
Speaker—Dr. Gaylord Anderson, University of Minnesota.

Afternoon

2.15 **Fort Garry Hotel.**
Chairman—Dr. F. G. McGuinness.

1. Obstetrical Topic:
Dr. Douglas E. Cannell, University of Toronto.

2. Management of the Menopause:
Round Table Conference.
Chairman—Dr. Elinor Black.

Tuesday, March 27th

Morning

St. Boniface Hospital.

9.00 **Clinical Program:**
X-ray Conference.
Management of Renal Disease.
Cough — as a Symptom.
Geriatric Topics.
Management of Diabetes.

Noon

12.15 **Luncheon, St. Boniface Hospital.**
Chairman—Dr. W. F. Abbott.
Speaker, Dr. Douglas E. Cannell, Professor of Obstetrics and Gynaecology, University of Toronto.

Afternoon

St. Boniface Hospital.
Chairman—Dr. D. S. McEwen.

2.15 **Hemorrhage in Obstetrical Practice:**
Dr. A. W. Andison.

Management of Thyroid Disease.
Round Table Conference.
Chairman—Dr. A. Hollenberg.

Wednesday, March 28th

Morning

Winnipeg General Hospital.

9.00 **The Rational Use of Quinidine:**
Dr. A. B. Houston.

Fatigue as a Symptom:
Dr. G. L. Adamson.

The Management of Anuria:
Dr. Ruben Cherniack.

The Sprue Syndrome, a Discussion of its Early Recognition:
Dr. D. L. Kippen.

10.40 **Surgical Topics:**
Dr. C. W. Burns and Staff.

Noon

12.15 **Luncheon, Winnipeg General Hospital.**
Nurses' Residence.
Speaker—Dr. Ray Farquharson, Professor of Medicine, University of Toronto.
Diagnosis and Treatment of Anaemias.

Afternoon

Medical College — Theatre "A"
Chairman—Dr. C. W. Burns.

2.15 **1. Palliation in Tumor Treatment:**
Dr. Walter C. MacKenzie, University of Alberta.

2. ACTH and Cortisone in Clinical Medicine:
Round Table Conference.
Chairman—Dean L. G. Bell.

3. Colles Fracture — Illustrated by Film:
Dr. F. Robert Tucker.

Thursday, March 29th

Morning

Deer Lodge Hospital.

9.00 **Clinico-Pathological Conference:**
Dr. J. D. Adamson, Dr. T. H. Williams and Staff.

Peripheral Vascular Disease:
Dr. C. E. Corrigan and Dr. L. R. Coke.

Modern Methods of Treatment of Hemiplegia:
Dr. W. M. Musgrove and Dr. John Matas.

Noon

12.15 **Luncheon, Deer Lodge Hospital.**
Chairman—Dr. W. R. Dunlop, Senior Treatment Medical Officer, Deer Lodge Hospital.
Speaker—Col. John N. Crawford, M.B.E., E.D., Medical Directorate, Army Headquarters, Ottawa.
The General Practitioner and Civil Defence.

Afternoon

Deer Lodge Hospital.

2.15 **1. The New Problems in Modern Warfare:**
Col. J. N. Crawford, M.B.E., E.D., Medical Directorate, Army Headquarters, Ottawa.

2. The Management of Gall Bladder Disease and Its Complications:
Round Table Conference.
Chairman—Dr. J. Wendell Macleod.

Evening

8.15 **Medical College.**

1. Intestinal Obstruction:
Dr. W. C. MacKenzie, Professor of Surgery, University of Alberta.

2. Medical Diseases of Bone:
Dr. Ray Farquharson, Professor of Medicine, University of Toronto.

Friday, March 30th

Morning

Children's Hospital.

9.00 **Clinical Program.**

Noon

12.15 **Luncheon, Children's Hospital.**
Chairman—Dr. Bruce Chown.
"Information Please"
A battery of experts will answer questions on any subject in pediatrics.

Afternoon

Children's Hospital.

2.00 **1. The Treatment of Some Common Skin Disorders in Children:**
Dr. Arthur R. Birt.

2. "Abdominal Surgical Emergencies in Children."
Round Table Conference.
Chairman—Dr. H. Medovy.

Evening

Dinner — Speaker to be announced.

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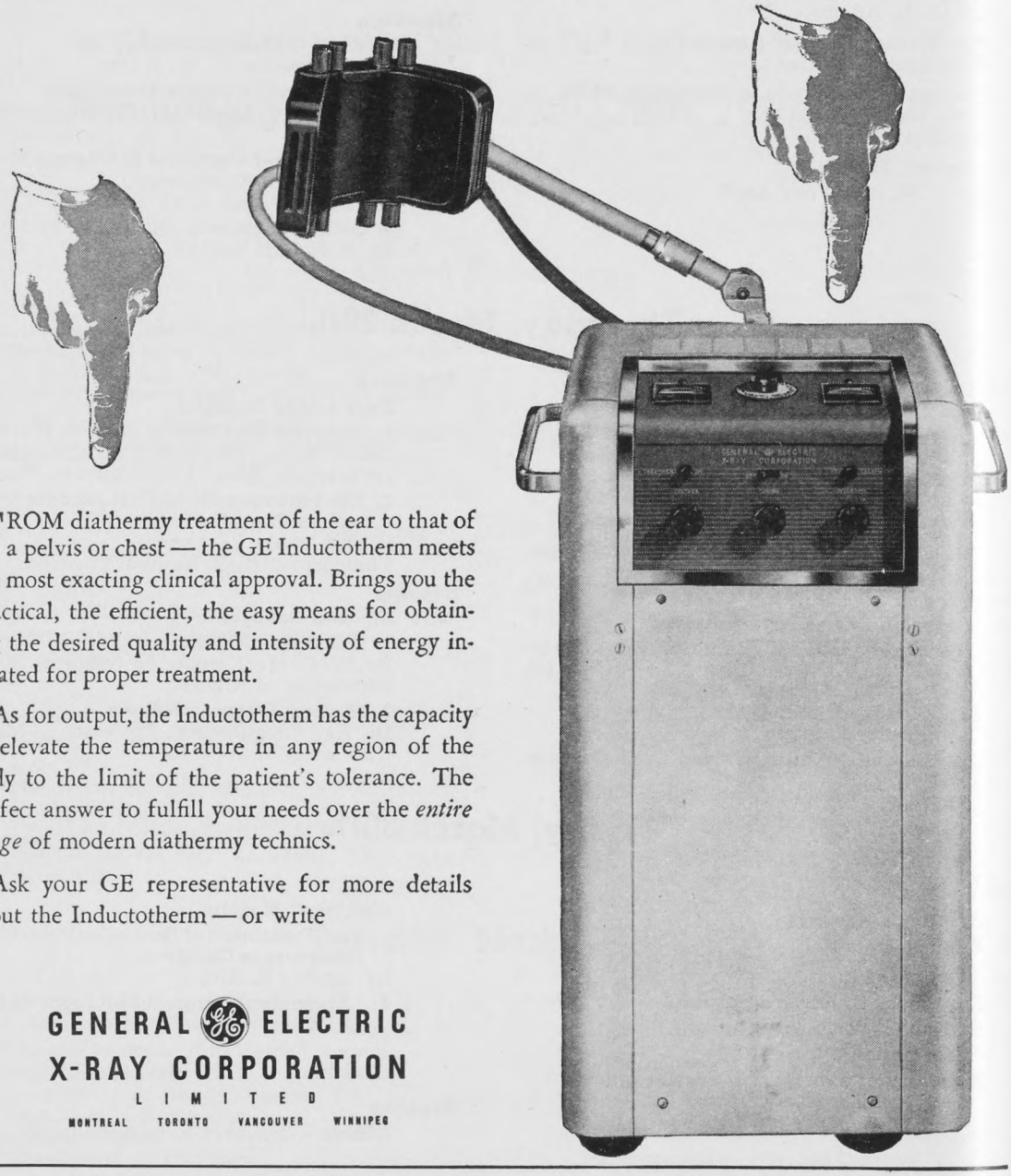

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EDITORIAL

J. C. Hossack, M.D., C.M. (Man.), Editor

March

We derive the present name of this month from the Romans among whom it was at an early period the first month of the year, as it continued to be in several countries to a comparatively late period, the legal year beginning even in England on the 25th of March till the change of the style in 1752. For commencing the year with this month there seems a sufficient reason in fact of its being the first season, after the dead of the year, in which decided evidences of a renewal of growth take place. And for the Romans to dedicate their first month to Mars and call it Martius seems equally natural, considering the importance they attached to war and the use they made of it.

Among our Saxon forefathers this month bore the name of Lenet—monat—that is, length month —in reference to the lengthening of the day at this season—the origin, also, of the term Lent.

Speechly and Culex

A few days ago when the mercury in the thermometer had almost completely crept into the little bulb in an effort to find warmth, there arrived a very timely booklet. It was the 1950 Report of the Anti-mosquito Campaign. Now if at any time of the year the thought of mosquitoes is tolerable it is when the temperature is thirty degrees below zero. Then in the mind's ear becomes audible a gentle hum. Imagination, on flimsy wings created by itself, carries us to that pleasant time when we can say, "The winter is past. The snow is over and gone. Flowers appear on the earth. The time of the singing of birds has come, and the voice of the turtle is heard in our land." And then, to spoil the picture, we remember that, blending with these more pleasing sounds, will be the thin voices of the mosquitoes.

That is, it used to be so. Now, thanks to Dr. Speechly, these disturbers of our peace, these minute provokers of great discomfort, no longer fill the air with their tinnient shrillings. No member of our profession has done so much to merit the goodwill and kindly feelings of his fellow-citizens as has Dr. Speechly. Because of his efforts they now can walk and recreate themselves on streets, in parks, in private arbours and new-planted gardens free from a nuisance always considered great and for long considered beyond control.

We are told by entomologists that only the females sting and, apparently, not all the females but only those which do not take part in the "characteristic dances." The ladies who sit out the dances, it seems, become blood addicts and this addiction leads to the ruination of their health and to their early decease. Unfortunately the poison does not act quickly and so they don't die early enough. Moreover it takes time (and blood) to develop the addiction, hence the need to take violent means to destroy both addicts and non-addicts. In the pre-Speechly days our solitary method was a vigorous slap which not infrequently missed its mark.

Twenty-five years ago Dr. Speechly declared war on the mosquito. Being a kindly man with a scientific mind he could appreciate its beauty and admire its wondrous and fragile structure. But, being human and generous, he was physically annoyed and morally shocked by the ingratitude of a creature which made a habit of biting the hands (and other parts) that fed it.

At first Speechly's was a voice crying in the wilderness; a voice, moreover, that was almost drowned by culexian trumpetings. He met with much skepticism, much indifference, little enthusiasm and almost no support. Such apathy and discouragement would have daunted many, but Speechly was dauntless. Even as Cato the Censor, in the palmy days of Rome, year after year, in season and out of season, declaimed "Delenda est Carthago"; so Speechly, in like fashion and with equal assiduity, cried "Delenda est Mosquito." And as it was with Cato and Carthage so has it been with Speechly and Culex.

How the little beggars must hate him. The front page of the booklet includes a portrait of Dr. Speechly with a gentle smile playing about his lips. If it were possible for a mosquito to (1) recognize a portrait and (2) quote from Shakespeare, it would be easy to conceive the idea of such an insect contemplating this picture of its arch-foe and moaning or shrieking "villain, villain, smiling damned villain." For verily as Saul slew his thousands, and David his hundreds of thousands of Philistines, so has Harry slain his thousands of millions of Children of Culex. He has smothered them in their cradles, has cut them down in the flower of their youth, and, in their age, has smitten them hip and thigh. He has fogged them with "Tifas," sprayed them with "Buffalos," crammed their bellies full of oil and D.D.T., and left the few survivors with anguished and despairing hearts. Thus, with a thoroughness and energy which U.N. must envy but dare not emulate, he has stopped aggression by extirpating the aggressors.

Viva Harry! Long live Speechly! To keep up the good work he wants $25,000. That's less than 10c per citizen and the best ten-cent's worth anyone can get anywhere.

ADVANCE *in antibiotic therapy*

for the first time this logical combination of antibiotics is available

Now, the combination of rapid-acting penicillin, repository penicillin and dihydrostreptomycin—in one convenient injection—places more effective therapy at the command of the physician. A broadened antibacterial spectrum gives greater coverage for more efficient and rapid control of many infections.

Penicillin S-R
Trade Mark

with Dihydrostreptomycin

Clinical Advantages of Penicillin S-R *with* Dihydrostreptomycin

- Effective against a wide range of gram-positive and gram-negative organisms
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indications: Infections due to organisms susceptible to penicillin and/or dihydrostreptomycin.

preparation and administration: PENICILLIN S-R with DIHYDROSTREPTOMYCIN is easy to prepare and inject. It does not plug needles as small as 20 or 21 gauge. To each single-dose vial aseptically add 2.2 cc. of: Water for Injection; Physiological Sodium Chloride Solution; or 5 per cent Dextrose Injection. Gently agitate to provide homogeneous suspension—solution for injection. A single dose (3 cc. prepared as directed) is injected intramuscularly, usually twice daily. Care must be taken to avoid intravenous injection, employing the usual precaution of aspiration.

Penicillin S-R *with* Dihydrostreptomycin

(Parke-Davis Penicillin and Dihydrostreptomycin Sulphate)

Each 3 cc. with aqueous diluent contains:

Crystalline penicillin-G procaine.....300,000 units
Crystalline penicillin-G sodium.....100,000 units
Dihydrostreptomycin (as the sulphate)....1.0 Gm.

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General Practitioners

General Practitioners' Association of Manitoba
In Affiliation with the Manitoba Medical Association

The General Practitioners' Prize

This is not one prize but two, to the value of one hundred and fifty dollars each, awarded annually by the General Practitioners' Association of Manitoba, to the final year medical student who is adjudged the best interne in each of two teaching hospitals and who has further stated his intention to enter general practice following graduation.

The prize—or prizes—was awarded for the first time in May, 1950. The worthy recipients were Doctor Kenneth B. Duncan who had interned in the Winnipeg General Hospital and Doctor Charles D. Lees who had spent his final year's interneship at St. Boniface Hospital. Doctor Lees commenced practice at Oak River, while Doctor Duncan is serving an additional post-graduate year's interneship before beginning practice this coming summer.

It is difficult to arrive at a decision as to just what qualities constitute the "best interne." This difficult decision is left to the judging committee. This committee is made up of the Dean of the Faculty of Medicine, the current President of the General Practitioners' Association and the Medical Superintendents of the two largest teaching hospitals, with power to add to their numbers if they wish.

It is generally agreed that the "best qualities" are not always found in the most brilliant student. Academic aptitude, while desirable, does not necessarily stamp one as a proficient interne. Diligence, for example, in the matter of keeping complete and orderly histories, and a meticulous attention to the many other hospital duties of an interne, are qualities much more to be desired than scholarship alone. We would venture the opinion that the top honours man each year will qualify as a winner in less than twenty-five per cent of future awards.

Should a final year student desire to advance himself with post-graduate training in order to make himself a better general practitioner, that is all to the good. It is the declared intention we want and we are not afraid that he will break faith at a later date. If we were, it would have been stipulated that the money would not be paid over until the prize-winner had actually begun general practice and such is Not the case.

So think it over you final year men and let's have your applications. After all have you ever reflected what a challenge it might be to a man if he should decide to become a "specialist in everything?"

Dr. Jack McKenty, First Vice-President.

College of Physicians and Surgeons of Manitoba

Registration Committee

November 6, 1950

Student Registration Granted

Peter George Premachuk, 3rd year, University of Ottawa.

Enabling Certificates Deferred

Grace Chow-Wei Chen, B.M., National Tung Chi U., 1945.

Kunigunda Zymantiene, M.D., U. Vytautas the Great, 1938; M.D., U. Munster, 1948.

Enabling Certificates Granted

Kye-ting King, B.Sc., St. John's U., 1938; M.D., St. John's U., 1941.

Chi-Kong Liu, M.D., National Central U., 1947.

Certificate of Registration Granted

George Henry Burgess, M.D., U. Tor., 1945; L.M.C.C., 1945.

Certificates of Licence Granted

Ovila Millette, B.A., U. Ottawa, 1944; M.D., Laval U., 1950; L.M.C.C., 1950.

Clement Lavoie, B.A., U. Man., 1941; M.D., Laval U., 1950; L.M.C.C., 1950.

Cecil Sorensen, B.A., U. Sask., 1947; M.D., U.W.O., 1949; L.M.C.C., 1949.

Robert Charles Elliott, M.D., C.M., Queen's U., 1944; L.M.C.C., 1944.

William James Taylor, M.B., Ch.B., U. Edin., 1947.

December 22, 1950

Student Registration Granted

Thadeus Zygmund Zwirkoski, 2nd year, University of Ottawa.

Enabling Certificate Granted

Chien-liang Hsu, B.Sc., Yenching U., 1928; M.D., Peiping Union Medical College, 1932.

Certificates of Registration Confirmed

Ta-Hui (David) Tien, M.D., Cheeloo U., 1944; L.M.C.C., 1950.

George White Allen, B.Sc., Walla Walla College, 1936; M.D., C.M.E., 1942; D.N.B., 1942; L.M.C.C., 1950.

Sheo-Nan Cheer, B.A., U. Nanking, 1917; M.D., Johns Hopkins U., 1920; D.N.B., 1921; L.M.C.C., 1950.

Shan-Ya Gin, M.D., Woman's Christian Medical College, 1931; D.C.H., R.C.P.S., London, 1948; L.M.C.C., 1950.

Ian Blake Thomson, M.B., Ch.B., U. Glasg., 1948.

Certificates of Registration Approved

Sheila Robertson Leyton, M.B., Ch.B., U. Aberd., 1943.

John Shaw Campbell, M.B., B.Ch., Queen's U. of Belfast, 1938; D.M.R.D., R.C.P.S., Eng., 1947.

SOCIAL NEWS

Reported by K. Borthwick-Leslie, M.D.

Congratulations to Dr. Ronald Jackson, son of Mr. and Mrs. Howard Jackson, Yorkton, Sask., who has been awarded the degree of Fellow of the Royal College of Surgeons, Edinburgh. Dr. Jackson graduated from Man. Medical College in 1944, served in the Navy for two years prior to his P.G. work. He is expected to return to Canada early in the spring.

♦

Dr. Robert Thomas Ross, 1948 Grad., son of Mr. and Mrs. John Ross, Scotia St., Winnipeg, is also to be congratulated on his appointment as member of the Royal College of Physicians, London, England. Dr. Ross has been doing post-graduate work in the National Hospital for Nervous Diseases, Queen St., London.

♦

Another appointment of interest to us all is that of Dr. A. B. Houston, B.Sc., F.R.C.P. (C), F.A.C.P., as Medical Director of the Monarch Life Assurance Co., Winnipeg. Working for our friend, Mr. Abbott, now A. B.?

♦

Dr. and Mrs. Henry Funk left by air early in February for Jamaica, for a wonderful holiday. They are expected back early in March.

♦

Dr. A. M. Campbell recently spent an extended holiday in Tampa, Florida, where he visited his son and wife, Dr. and Mrs. Robert Campbell, and became re-acquainted with those four grandchildren. Dr. "Bob" is a Manitoba graduate of 1937, and enjoying life in Florida.

♦

Welcome back into professional circulation to Dr. Washington. He looks wonderful. Apparently even enforced holidays are good for us.

♦

Dr. and Mrs. Donald C. Brereton announce the arrival of William Donald, February 6th.

♦

Dr. and Mrs. Lyle Henry (nee Betty Robson) are happy to announce the birth of Barbara Gail, February 9th.

♦

Dr. and Mrs. J. C. McCawley announce the birth of Deborah Joan, February 2nd.

♦

Born at Flin Flon, Man., on February 9th, to Dr. and Mrs. Glen Willson, a son, David Lee, brother for Mark.

Dr. and Mrs. W. McKinnon are happy to announce the arrival of Marilyn Dianne, on February 2nd. Brian is also very happy about his baby sister.

♦

Dr. and Mrs. D. W. Hunt, 944 Windermere Ave., Fort Garry, announce the birth of their third son, Patrick Wm. David. Quotation from my son David, "Gee, Mom, isn't it a good thing the stork doesn't get mixed up like the postman at Xmas." Explanation: 944 Sommerville next street over, spends a lot of time at Xmas returning mail addressed to Windermere. For sure I agree with David!

♦

Sincere sympathy to N.S. (Matron) Edna Leishman and Dr. John, of Regina, on the loss of their father, Dr. Leishman.

♦

Dr. and Mrs. Bjorn Jonsson of Baldur, Man., are happy to announce the arrival of Randver Fitzgerald, brother to Jon, on February 9th, 1951.

♦

Dr. John Hillsman has just returned from an airmail jaunt to New Haven and New York where he attended a farewell banquet in honor of Prof. S. C. Harvey, on his retirement from the active teaching staff at Yale. Dr. Hillsman and the newly appointed professor of surgery, Dr. Lindskog, were the guest speakers, as John says, "One clean, one dirty"—use your own judgment. Dr. John met his son Dean, who was taking part in the Yale-Dartmouth swimming meet, which unfortunately Dartmouth lost by a narrow margin. Except for a tough trip home because of "Sea Food Enteritis" it was an extremely pleasant, brief holiday.

♦

Gordon, may I refer you to Ciba's most recent "Therapy" Pamphlet? Pages 28 or 21? Or would it be more appropriate to refer all your authors and editors?*

♦

*So-**she**-ell Ed.—Do you not know that reading Direct Mail literature is a bitter potion for me to swallow? If Dave Menzies of Ciba, will insert said beneficial information in his Review advertising, I, like all Review readers, will digest it thoroughly. Should there be a ray of hope, free samples will be requested. G.

B-OMNYL

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Beriberi, latent beriberi; anorexia of dietary origin; conditions of increased metabolism such as febrile conditions and hyperthyroidism; pellagra either mild or acute.

Each teaspoonful contains

HIGH POTENCY

Thiamine Hydrochloride . .	2 mg.
Riboflavin	1 mg.
Niacinamide	10 mg.
Pyridoxine Hydrochloride .	.5 mg.
Calcium d-Pantothenate . .	2.5 mg.

Supplied in 8 oz.—16 oz. and 80 oz. bottles.

Vitamin B-Complex as derived from 0.33 grams of Rice Bran Extract and Yeast Extract per millilitre with added Thiamine, Riboflavin, Niacinamide, Pyridoxine Hydrochloride and Calcium d-Pantothenate.

B-OMNYL is available in capsule form in bottles of 100 and 500.

CHARLES R. WILL & COMPANY LIMITED

LONDON — CANADA

"ETHICAL PHARMACEUTICALS"

Professional Representative: Mr. J. R. Hope, 264 Lindsay Street, Winnipeg, Man.

ASSOCIATION PAGE

Reported by M. T. Macfarland, M.D.

Excerpts from Food and Drugs Act

"drug" includes all medicine for internal or external use for man or animal; any substance, mixture of substances and any article that may be used for the diagnosis, treatment, mitigation or prevention of disease in man or animal; any cosmetic; any material that may be used for disinfection in premises in which food is manufactured, prepared or kept or for the control of vermin in such premises; (Section 2, Subsection C)

"medicine" means any substance or mixture of substances that may be used in restoring, correcting or modifying organic functions; (Section 2, Subsection J)

No person shall import, offer for sale, or sell any food or drug represented by label or by advertisement to the general public as a treatment for any of the diseases, disorders or abnormal physical states named or included in Schedule A to this Act or in any amendment to such Schedule; (Section 6A)

Schedule A

Alcholism, Appendicitis, Arteriosclerosis, Blood Poisoning, Bright's Disease, Cancer, Diabetes, Diphtheria, Disorders of menstrual flow, Disorders of the prostatic gland, Dropsy, Epilepsy, Erysipelas, Gallstones, Kidney Stones, Bladder Stones, Gangrene, Goitre, Heart Disease, High Blood Pressure, Infantile Paralysis, Influenza, Lockjaw, Locomotor Ataxia, Obesity, Pleurisy, Pneumonia, Ruptures, Scarlet Fever, Sexual Impotence, Small Pox, Spinal Meningitis, Trachoma, Tuberculosis, Tumors, Typhoid Fever, Ulcers of the gastro-intestinal tract, Venereal Diseases.

"prescription" means a written order issued and signed by any person authorized to treat patients with drugs in any province of Canada directing the dispensing of a stated amount of any drug or mixture of drugs to the patient named in such order. (A.02.016)

No person shall sell a drug or a preparation containing a drug named or included in Appendix IV (1) except on prescription(2), nor shall any person refill such prescription unless the prescriber thereof so directs in writing thereon. (C.01.016).

Notwithstanding the provisions of C.01.016 a drug or a preparation containing a drug named or included in Appendix IV may be sold without a prescription to a (a) physician, (b) dentist, (c) veterinary surgeon, (d) drug manufacturer, (e) wholesale druggist, or (f) retail druggist. (C.01.017).

Appendix IV

Prescription Drugs

*Adrenocorticotrophic Hormone, A.C.T.H.

Aminopyrine and any salt, homologue, or derivative thereof

Amphetamine and any salt thereof

Aureomycin and any salt or derivative thereof

Barbituric acid and any salt, homologue, or derivative thereof

Cinchophen and Neocinchophen

*Cortisone

d-desoxyephedrine and any salt thereof

Methedrine and any salt thereof

Ortho-dinitrophenol and any compound, homologue, or derivative thereof

Penicillin, its salts or derivatives, or preparations thereof, excluding preparations for oral use that contain not more than 3,000 International Units per dose

Pervitin and any salt thereof

Phenytoin Sodium

Streptomycin and any compound thereof

Sulphonamides and any salt, homologue, or derivative thereof

Tetraethylthiuram disulphide

Thiouracil and any homologue, or derivative thereof

Thyroid

Thyroxin and any salt thereof

Urethane

*(Amendment P.C. 6225, 28 December, 1950, Canadian Gazette 10/1/51).

Survival Under Atomic Attack

Extract From the Official United States Government Booklet

(Executive Office of the President, National Security Resources Board, Civil Defense Office, NSRB Doc. 130).

You can survive. You can live through an atom bomb raid and you won't have to have a Geiger counter, protective clothing, or special training in order to do it. The secrets of survival are:

Know the bomb's true dangers.

Know the steps you can take to escape them.

Kill the Myths

Atomic weapons will not destroy the earth. Atomic bombs hold more death and destruction than man ever before has wrapped up in a single package, but their over-all power still has very definite limits. Not even hydrogen bombs will blow the earth apart or kill us all by radioactivity.

Doubling bomb power does not double destruction. Modern A-bombs can cause heavy damage 2 miles away, but doubling their power would extend that range only to 2½ miles. To stretch the damage range from 2 to 4 miles would require a weapon more than 8 times the rated power of present models.

Radioactivity is not the bomb's greatest threat. In most atom raids, blast and heat are by far the greatest dangers that people must face. Radio-

activity alone would account for only a small percentage of all human deaths and injuries, except in underground or underwater explosions.

Radiation sickness is not always fatal. In small amounts, radioactivity seldom is harmful. Even when serious radiation sickness follows a heavy dosage, there is still a good chance for recovery.

Six Survival Secrets for Atomic Attacks

Always put first things first; never lose your head.

1. Try to get shielded. If you have time, get down in a basement or subway. Should you unexpectedly be caught out-of-doors, seek shelter alongside a building, or jump in any handy ditch or gutter.

2. Drop flat on ground or floor. To keep from being tossed about and to lessen the chances of being struck by falling and flying objects, flatten out at the base of a wall, or at the bottom of a bank.

3. Bury your face in your arms. When you drop flat, hide your eyes in the crook of your elbow. That will protect your face from flash burns, prevent temporary blindness and keep flying objects out of your eyes.

4. Don't rush outside right after a bombing. After an air burst, wait a few minutes, then go help to fight fires. After other kinds of bursts wait at least 1 hour to give lingering radiation some chance to die down.

5. Don't take chances with food or water in open containers. To prevent radioactive poisoning or disease, select your food and water with care. When there is reason to believe they may be contaminated, stick to canned and bottled things if possible.

6. Don't start rumors. In the confusion that follows a bombing, a single rumor might touch off a panic that could cost your life.

Five Keys to Household Safety

1. Strive for "fireproof housekeeping." Don't let trash pile up, and keep waste paper in covered containers. When an alert sounds, do all you can to eliminate sparks by shutting off the oil burner and covering all open flames.

2. Know your own home. Know which is the safest part of your cellar, learn how to turn off your oil burner and what to do about utilities.

3. Have emergency equipment and supplies handy. Always have a good flashlight, a radio, first-aid equipment and a supply of canned goods in the house.

4. Close all windows and doors and draw the blinds. If you have time when an alert sounds, close the house up tight in order to keep out fire sparks and radioactive dusts and to lessen the chances of being cut by flying glass. Keep the house closed until all danger is past.

5. Use the telephone only for true emergencies. Do not use the phone unless absolutely necessary. Leave the lines open for real emergency traffic.

Atom Splitting is Just Another Way of Causing an Explosion

To begin with, you must realize that atom-splitting is just another way of causing an explosion. While an atom bomb holds more death and destruction than man has ever before wrapped in a single package, its total power is definitely limited. Not even hydrogen bombs could blow the earth apart or kill us all by mysterious radiation.

Your Chances of Surviving an Atomic Attack Are Better Than You May Have Thought

Because the power of all bombs is limited, your chances of living through an atomic attack are much better than you may have thought. In the city of Hiroshima, slightly over half the people who were a mile from the atomic explosion are still alive. At Nagasaki, almost 70 per cent of the people a mile from the bomb lived to tell their experiences. Today thousands of survivors of these two atomic attacks live in new houses built right where their old ones once stood. The war may have changed their way of life, but they are not riddled with cancer. Their children are normal. Those who were temporarily unable to have children because of the radiation now are having children again.

To Sum Up

To sum up, always remember that blast and heat are the two greatest dangers you face. The things that you do to protect yourself from these dangers usually will go a long way toward providing protection from the explosive radioactivity loosed by atomic explosions.

While the lingering radioactivity that occasionally follows some types of atomic bursts may be dangerous, still it is no more to be feared than typhoid fever or other diseases that sometimes follow major disasters. The only difference is that we can not now ward it off with a shot in the arm; you must simply take the known steps to avoid it.

Keep Your Head. Don't touch off a panic that may cost your life. If you follow the pointers in this article, you stand far better than an even chance of surviving the bomb's blast, heat, and radioactivity. What's more, you will make a definite contribution to civil defense in your community, because civil defense must start with you. But if you lose your head and blindly attempt to run from the dangers, you may touch off a panic that will cost your life and put tremendous obstacles in the way of your Civil Defense Corps.

Civil Defense in Manitoba

There is little to report of specific Medical interest at this date in regard to Civil Defense in Manitoba. A recent meeting of the Winnipeg Medical Society included some discussion of the medical phases of the problem and afforded an opportunity for the introduction of Mr. A. C.

Delaney who has recently been appointed "Provincial Civil Defense Officer."

The booklet "Organization for Civil Defense," published in October, 1950, under the authority of the Minister of National Defense gives the Civil Defense organizational charts at the Federal and Provincial level. A basic understanding of them is necessary before the responsibilities of local groups can be understood.

At the Federal level Authority flows from cabinet to cabinet defense committee to Minister of National Defense. The latter is advised by: (1) "Dominion-Provincial standing committee on Civil Defense" made up of Federal and Provincial Ministers responsible for Civil Defense, (2) "Civil Defense co-ordinating committee," made up of representatives from related Federal Departments and Services. The key individual at the Federal level—responsible directly to the Minister of National Defense is the "Co-ordinator of Civil Defense," Major-General F. F. Worthington.

At the Provincial level authority flows from government to Cabinet to the Minister responsible for Civil Defense—Honourable Mr. C. Rhodes Smith, K.C., Provincial Attorney-General. For advice this Minister has set up a "Provincial Civil Defense planning Committee" with representatives from Departments of Government and certain other closely related agencies such as St. John Ambulance and Canadian Red Cross. Dr. M. T. Macfarland is a member of this committee representing the Manitoba Medical Association. Dr. Hugh Malcolmson has been designated as the representative of the Health Section of the Provincial Department of Health and Public Welfare. Directly responsible to Mr. Smith is Mr. A. C. Delaney, full time "Provincial Civil Defense Officer."

Below this level all activity rests with the local community. Greater Winnipeg has its own "Civil Defense Control" Committee and "Civil Defense Planning Committee." At this time, attempts are being made to acquire the full time service of a "Civil Defense Officer." His will be the task of working out the problems and the development of well rounded Civil Defense services. The physicians who will work at this level have not as yet been designated.

To date the provincial committee has held one meeting where broad principles were discussed and when Mr. Smith introduced Mr. Delaney to the group at large. It was agreed that all communities of Manitoba had their part to play in the Civilian Defense effort.—Hugh Malcolmson, M.D.

General Council, C.M.A.

The following letter was sent to the District Medical Societies and Sections of the Association:

"The Annual Meeting of the Canadian Medical Association will be held at Montreal on June 18th to 22nd, 1951. Convention Headquarters will be at the Mount Royal Hotel.

The Manitoba Division is entitled to name eight members to General Council in addition to the President and Secretary, or Joint Secretaries.

Will you kindly advise which members of your group are planning to attend the Montreal meeting and would be eligible for selection to General Council.

Your early attention to this matter will be appreciated."

Medical Care for Welfare Cases

In the local legislature recently one of the C.C.F. members gave notice of motion for a proposed resolution that the government should give consideration to the advisability of providing a supplementary allowance, based on the cost of living, to the old age and blind pensioners of Manitoba, and also providing free medical, dental and other health services to our old and blind pensioners. Since there was little promised support for the resolution it was withdrawn by the sponsor.

Sera Gratis

Attention of all members is drawn to the section of the department of Health and Public Welfare which outlines the products which are available without charge to the physician for immunization procedures and the treatment of certain conditions for which the department assumes responsibility. Each member should be familiar with the list of materials and he should make certain that the charge for administration should not include the cost of the sera concerned. Likewise if the sera is purchased by the doctor and utilized for a Workmen's Compensation Board case no allowance is made for the material by the latter since it is available to all citizens of the province without charge.

Requests for Sterilization

The Annual Report of the Medical Defence Union (England) which was presented to the General Meeting of the Union on September 20, 1949, has this comment concerning requests for sterilization.

"It is mentioned that members are from time to time approached by patients with a request for sterilization. Counsel's opinion on this matter was first sought some years ago, but owing to the time which has elapsed since then, and the changes in medical thought and social conscience, it was felt that a new opinion should be taken. The opinion is that sterilization on eugenic grounds alone is illegal, but that it is not so if there are valid therapeutic reasons—in other words, if there are conditions which may imperil health or life if steriliztion is not performed. This places sterilization on the same basis as abortion and calls for similar safeguards (including a second opinion and the written consent of the patient) on the part of the practitioner."

M. T. M.

America Academy of General Practice

The 1951 Scientific Assembly of the Academy will be held at San Francisco, California, from March 19 to 22.

The programme has been keyed on the general practitioner's role of family physician. Canadian physicians will be most welcome.

Announcement folders may be secured through Mr. Mac F. Cahal Broadway at 34th St., Kansas City, Mo.

Clinical Luncheons

Time Table for Clinical Luncheons held during the Season in Greater Winnipeg Hospitals. The days in each month on which the luncheons are held are listed herewith. Visiting doctors are welcome.

1st Monday—Deer Lodge Hospital.
1st Thursday—Winnipeg General Hospital.
1st Friday—Children's Hospital.
2nd Tuesday—Municipal Hospital.
2nd Tuesday—Misericordia Hospital.
2nd Thursday—St. Boniface Hospital.
2nd Friday—Victoria Hospital
3rd Tuesday—Grace Hospital.
3rd Thursday—Winnipeg General Hospital.
4th Tuesday—St. Joseph's Hospital.
4th Thursday—St. Boniface Hospital.

Anaesthesiology Section

1st Tuesday—Regular meetings of the Anaesthesiology Section of the Winnipeg Medical Society. Visiting anaesthetists are welcome.

Directory

Names and Phone Numbers of

Detailmen

Representing Review Advertisers in this issue, whose names are not listed under a business address.

Abbott Laboratories
G. J. Bowen 44 559
R. G. (Bud) Harman 592 648
D. A. Tedford 724 863

Allen & Hanburys Co.
H. W. Heaslip 39 401
E. M. Tackaberry 404 184

Ayerst, McKenna and Harrison
W. R. Card 49 829
C. G. Savage 34 558

British Drug Houses
F. J. Burke 34 413

Ciba Company Ltd.
J. T. Dewar 402 540
Fred Ruppel 63 158

Connaught Laboratories
Brathwaites Ltd. 922 635

Frosst, Chas. E.
W. M. Lougheed 403 963
W. J. McGurran 208 231
E. R. Mitchell 402 132

General Electric X-ray Corp.
W. A. Martin 924 277

Horner, Frank W.
Gordon Couch 404 920
George D. Craine 201 822
Cam Loptson 48 135

Mead Johnson
George Moore 404 0

Nestle's Milk Products
Herb. Harvey 724 5

Ortho Pharmaceutical Corp.
J. G. Johnson 928 6

Park, Davis & Co.
L. W. Curry 401 1
B. S. Fleury 404 3

Poulenc Limited
W. J. Plumpton 55 6

Sandoz Pharmaceuticals Ltd.
H. D. Robins 39 9
G. A. Roddick 928 6

Schmid, Julius
E. E. Conway 64 2

Shuttleworth, E. B.
G. D. Roddick 596 0

Squibb & Son, E. R.
MacArthur, J. H. Don 404 7

Will, Chas R.
John R. Hope 401 8

Winthrop-Stearns
Geo. Edmonds 49 7
R. M. Kelly 34 5

Wyeth & Bro., John
A. W. Cumming 35 2
W. J. Tarbet 423 4

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1950 Jan. 1 to Jan. 27,'51	1950 Dec. 3 to Dec. 30,'50	1949 Jan. 1 to Jan. 28,'50	1949 Dec. 4 to Dec. 31,'49
Anterior Poliomyelitis	0	0	0	3
Chickenpox	176	219	172	204
Diphtheria	1	0	2	1
Diarrhoea and Enteritis, under 1 yr.	5	4	1	8
Diphtheria Carriers	0	0	0	0
Dysentery—Amoebic	0	0	0	0
Dysentery—Bacillary	1	0	0	1
Erysipelas	1	1	3	1
Encephalitis	0	0	0	0
Influenza	4	3	2	13
Measles	326	214	147	268
Measles—German	3	0	0	1
Meningococcal Meningitis	0	2	2	0
Mumps	148	113	26	10
Ophthalmia Neonatorum	0	0	0	0
Pneumonia—Lobar	17	21	4	18
Puerperal Fever	0	0	0	0
Scarlet Fever	60	37	28	48
Septic Sore Throat	4	1	5	0
Smallpox	0	0	0	0
Tetanus	0	0	0	0
Trachoma	0	0	0	0
Tuberculosis	29	83	38	3
Typhoid Fever	0	0	0	5
Typhoid Paratyphoid	0	1	0	0
Typhoid Carriers	0	0	0	0
Undulant Fever	0	3	0	1
Whooping Cough	24	97	5	11
Gonorrhoea	114	99	97	87
Syphilis	12	12	24	29

Four-Week Period, December 31st to January 27th, 1951

DISEASES (White Cases Only) *Approximate population.	*779,000 Manitoba	*861,000 Saskatchewan	*3,825,000 Ontario	*2,952,000 Minnesota
Anterior Poliomyelitis	....	2	3	1
Chickenpox	176	438	2813	....
Diarrhoea & Enteritis (under 1 yr.)	3	....	....	....
Diphtheria	1	3	5	8
Diphtheria Carriers	....	....	....	....
Dysentery—Amoebic	....	....	....	5
Dysentery—Bacillary	1	....	7	....
Encephalitis Epidemica	....	....	1	....
Erysipelas	1	2	2	....
Influenza	4	....	73	2
Jaundice, Infectious	....	15	14	....
Measles	325	98	7518	299
German Measles	3	46	587	....
Meningitis Meningococcal	....	1	11	8
Mumps	148	741	2185	....
Ophthalmia Neonatorum	....	....	....	....
Pneumonia, Lobar	17	....	....	....
Puerperal Fever	....	....	....	....
Scarlet Fever	60	65	225	101
Septic Sore Throat	4	3	18	22
Smallpox	....	....	....	....
Tetanus	....	....	....	....
Trachoma	....	....	....	....
Tuberculosis	29	17	89	31
Trichinosis	....	....	....	....
Tularemia	....	....	....	....
Typhoid Fever	....	....	3	1
Typhoid Para-Typhoid	....	....	....	....
Typhoid Carrier	....	....	....	....
Undulant Fever	....	....	7	14
Whooping Cough	23	14	440	94
Gonorrhoea	114	....	186	....
Syphilis	12	....	83	....

*DEATHS FROM REPORTABLE DISEASES

For the Month of January, 1951

Urban — Cancer, 50; Influenza, 3; Measles, 1; Pneumonia, Lobar (108, 107, 109), 6; Pneumonia (other forms), 6; Pneumonia of newborn, 1; Syphilis, 2; Tuberculosis, 7; Neoplasms of Lymphatic and Haematopoietic Tissues, 3; Benign Neoplasms, 2; Gastro-Enteritis and Colitis, 2. Other deaths under 1 year, 19. Other deaths over 1 year, 215. Stillbirths, 15. Total, 249.

Rural—Cancer, 22; Diphtheria, 1; Influenza, 4; Pneumonia, Lobar (108, 107, 109), 3; Pneumonia (other forms), 12; Syphilis, 1; Tuberculosis, 4; Whooping Cough, 1; Neoplasms of Lymphatic and Haematopoietic Tissues, 3; Gastro-Enteritis and Colitis, 1. Other deaths under 1 year, 17. Other deaths over 1 year, 163. Stillbirths, 8. Total, 188.

Indians—Pneumonia (other forms), 2; Bacillary Dysentery, 1. Other deaths over 1 year, 2. Total 2.

*As reported to date.

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BIOLOGICS SUPPLIED FREE

Once again we wish to remind the physicians in Manitoba that the following are supplied free of cost by the Manitoba Department of Health and Public Welfare, Section of Preventive Medical Services, 320 Sherbrook Street, Winnipeg:

Smallpox Vaccine.
Diphtheria Toxoid.
Diphtheria Toxoid with Pertussis Vaccine (Combined).
Diphtheria Toxoid with Pertussis Vaccine and Tetanus Toxoid.
Diphtheria Toxoid with Tetanus Toxoid.
Pertussis Vaccine.
Tetanus Toxoid.
Typhoid Paratyphoid Vaccine.
Typhoid Paratyphoid Vaccine, with Tetanus Toxoid.
Scarlet Fever Toxin.
Schick Tests.
Dick Tests.
Diphtheria Antitoxin.
Scarlet Fever Antitoxin.
Tetanus Antitoxin.
Silver Nitrate Solution 1%.
Vollmer Patch Tests for Tuberculin Testing.

As these are supplied free of cost they are **not to be sold** under any circumstances (see Public Health Act, Section 34 (4).

MEDICAL LIBRARY

The University of Manitoba, Faculty of Medicine

The Medical Library will prepay all postage charges on Rural Manitoba Loans. There is therefore no charge to the Borrower either when the Loans are sent or returned.

Recent Accessions

General List

Thorek, Max. Modern surgical technic.
Lippincott, 1938.

Thorn, G. W. The diagnosis and treatment of adrenal insufficiency.
Blackwell, 1949. 171 p.

Thorner, M. W. Psychiatry in general practice.
Saunders, 1948. 659 p.

Tidy, Sir Henry L. A synopsis of medicine. 9th ed.
Wright, 1949. 1243 p.

Tobias, Norman. Essentials of dermatology. 3rd ed.
Lippincott, 1948. 518 p.

Tracey, M. V. Proteins and life.
Pilot Press, 1948. 154 p.

Trueta, Josep. An atlas of traumatic surgery illustrated histories of wounds of the extremities.
Blackwell Scientific Publication, 1949. 150 p.

Tuft, Louis. Clinical allergy. 2nd. ed.
Lea and Febiger, 1949. 690 p.

Van Rooyen, C. E. and Rhodes, A. J. Virus diseases of man.
Nelson, 1948. 1202 p.

Walker, A. E. Post traumatic epilepsy.
Thomas, 1949. 86 p.

Whipple, G. H. Hemoglobin, plasma protein and cell protein.
Thomas, 1948. 27 p.

Willard, H. H., Merritt, L. L. and Dean, J. A. Instrumental methods of analysis.
Van Nostrand, 1949. 247 p.

Winsbury-White, H. P. Textbook of genito-urinary surgery.
Livingstone, 1948. 1046 p.

Woerdeman, M. W. and Raven, C. P. Experimental embryology in the Netherlands, 1940-1945.
Elsevier, 1946. 132 p.

Work, T. S. and Work, Elizabeth. The basis of chemotherapy.
Oliver and Boyd, 1948. 435 p.

World Health Organization. Manual of the international statistical classification of diseases, injuries and causes of death. 6th rev.
The Organization, 1948.

Yater, W. M. Fundamentals of internal medicine. 3rd ed.
Appleton-Century, 1948. 1451 p.

Continuations

Conference on metabolic inter-relations. Transactions, 1949.

Text Books and Reference Text Books on Restricted Loan During the College Year

Andrews, G. C. Diseases of the skin for practitioners and students. 3rd ed.
Saunders, 1946. 937 p.

Bailey, Hamilton. Demonstrations of physical signs in clinical surgery. 11th ed.
Wright, 1949. 426 p.

Best, C. H. and Taylor, N.B. The physiological basis of medical practice. 5th ed.
Williams, 1950. 1330 p.

Diseases of women, by ten teachers, ed. by C. White, F. Cook and Sir William Gilliatt. 8th ed.
Arnold, 1948. 461 p.

Evans, C. A. L. Principles of human physiology. 10th ed. Orig. written by E. H. Starling.
Churchill, 1949. 1193 p.

Fulton, J. F., ed. A textbook of physiology. 16th ed.
Saunders, 1949. 1258 p.

Illingworth, C. F. W. A short textbook of surgery. 4th ed.
Churchill, 1947. 680 p.

Karsner, H. T. Human pathology. 7th ed.
Lippincott, 1949. 927 p.

Walshe, F. M. R. Diseases of the nervous system described for practitioners and students. 6th ed.
Livingstone, 1949. 359 p.

Wiggers, C. J. Physiology in health and disease. 5th ed.
Lea & Febiger, 1949. 1242 p.